CONSIDERATIONS IN ANAESTHESIA AND RESUSCITATION

By

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DEDICATION

This Book is dedicated to my professors of anaesthesia who have taught me how to deal with the patient in a scientific and a human manner and to my wife who always supports me willingly and happily.

PREFACE

Readers are advised to review anatomical, physiological, and pharmacological considerations for anaesthesia and resuscitation to get the utmost benefit of the coming pages that can be read within few days.

M.E. MOEMEN

I — CONSIDERATIONS IN PRACTICE OF ANAESTHESIA



PRE-OPERATIVE MANAGEMENT OF THE SURGICAL PATIENT

PRE-OPERATIVE EXAMINATION

It is logic that no patient may receive an anaesthetic, whether general or local without being, clinically examined. The anaesthetist should read the patients notes and pay attention to his investigations. In addition, he may ask for other investigations which should be performed even in an emergency surgery, if he feels indispensible. It should be put in mind that every patient pays a price for his anaesthesia that may be either one cell or his life. So, a careful examination of the patient together with a good history taking of previous diseases, anaesthetics or medicaments are the first duty of the anaesthetist.

The anaesthetist is always confronted with patients of different varieties. Some of them are fit adults while others may be old, young or with bad general condition. Some patients may need full doses of anaesthetic drugs while others may need smaller doses. Strangely enough children need larger dosage in comparison with their size, due to hyper-active metabolism. The careful pre-anaesthetic examination will allow the anaesthetist to design a successful anaesthetic plan as he considers all informations related to the particular patient to tide him over a particular surgery.

The anaesthetist should assess the efficiency of the respiratory and circulatory systems. So, he should ask the patient about

cough, sputum, dyspnoea on moderate exertion, orthopnoea, history of retrosternal pain or swelling of the ankles. He should then examine the patient for congested neck veins, central or peripheral cyanosis, clubbing of fingers, oedema of lower limbs, functional or organic cardiac lesions, cardiac arrhythmias, crepitations at the lung bases and enlargement of the liver. A heart which is failing or with a history of failure, cannot thus be missed. The arterial blood pressure, the haemoglobin concentration in the blood and the albumin and sugar in urine should be estimated. Informations about other systems i.e. hepatorenal should be considered. Some informations that seem minor may save the anaesthetist much trouble. The accessibility of peripheral veins, the position of the trachea in the neck and the presence of loose or artificial teeth are but few examples. The clever anaesthetist always asks the patient about previous allergic diseases or previous sensitivity to a particular drug.

In deciding the patient's fitness to withstand a certain surgical procedure, the anaesthetist should balance the urgency and severity of surgery against the state of the patients health. Usually a patient who carries out his daily work without distress or that who climbs a flight of stairs without marked dyspnoea is fit for an operation. Some patients, in the fifth decade of their life may present with a mild degree of hypertension that may only need a few days bed-rest with salt restriction, minimal sedatives or antihypertensive drugs before the operation. Patients with chronic anaemia should receive a transfusion of whole blood, or better, of packed red cells before the operation to increase the

oxygen carrying capacity of their blood. Patients with emphysema, chronic bronchitis or bronchial asthma may present certain problems. There is no hard and fast rule but the anaesthetist should judge each patient on his merits and if in doubt as to the presence or significance of any physical sign, he should not hesitate to call upon the consultation of a physician. When the anaesthetist studies enough medical bases and becomes indulged in anaesthetic training, supervised by senior anaesthetists, he will know how to manage patients of specific problems as diabetes, thyrotoxicosis, hypertension or myasthenia gravis. He will know how to solve the problems of patients presenting with drug intake as corticosteroids, diuretics or beta-blockers. He will get the training of specific techniques as induced hypotension or total intravenous anaesthesia where the theoretical basic sciences go hand in hand with the practical application in the mysterious attractive art of anaesthetic practice.

PRE-OPERATIVE PREPARATION

Pre-operative preparation of the surgical patient aims to enable him to meet anaesthetic and operative stresses with the least physiological and psychological disturbances.

For physiological preparation, the anaesthetist should aim at correction of possibly three deficits in his patient, namely : concentrational, volume or caloric deficits.

Concentrational deficit :

In acute blood loss, patients lose both haemoglobin and plasma. They expand their plasma volume to compensate for the loss of red cell mass. This may be manifested as tachycardia with or without hypotension. On the other hand, patients with chronic anaemia have normal total blood volumes because they adapt by expansion of their plasma volume to supernormal levels and they have normal pulse rate and blood pressure.

Before a surgical operation ,concentrational deficits in red cell mass should be corrected. In acute blood loss, whole blood should be transfused. In chronic anaemia packed red cells are transfused to save the kidney the burden of excreting more plasma as urine. In both conditions, the anaesthetist aims at a minimum haemoglobin concentration of 10 gm/100 ml of blood, to ensure a suitable blood oxygen carrying capacity.

Volume deficit :

Blood loss may be visible as in frank bleeding or as a swelling accompanying fracture femur. Fluid loss may be estimated in a third degree burn. On the other hand, fluid loss is sometimes concealed, a third space loss, as in fairly long standing vomiting or diarrhoea before hospitalization of the patient. In such circumstances the loss may be accompanied by electrolyte imbalance. Fluid loss may be also associated with a concentrational deficit.

Not all volume and concentrational deficits need to be corrected before operation, but a significant fraction of the total deficits should be corrected for the patient safety during anaesthesia and surgery. Volume deficits should be corrected depending on the clinical condition of the patient i.e. pulse, blood pressure and urine output, together with other parameters as haemoglobin concentration and appearance of skin and mucous membranes. The general condition of the patient including his mental state, his cardiovascular system, kidney function and body temperature should be also taken in consideration. Electrolyte and concentrational deficits associated with volume deficits should be corrected in guidance with specific laboratory estimations.

Caloric deficit :

A patient submitted to an operation may show a caloric deficit. A long standing restriction from diet and fluid intake, the pharmacologic effects of preoperative drugs and preoperative purgatives or laxatives, may impose dangerous metabolic stresses in the form

of depletion of stored glycogen, inability of the heart to increase its output, inability of the liver to produce adequate protein, respiratory insufficiency, and even inadequate kidney function.

So, the correct and desired pre-operative preparation of the surgical patient should aim at a state of an abundant nutritional intake. Patients should never be starved and food is only restricted for 6-8 hours and fluid for only 4 hours before operation. Purgatives and laxatives should not be allowed except before operations calling for previous emptying of the bowel.

The problem of the malnourished patient needs a solution. Such a patient goes with depleted fuel stores. His loss of muscle mass makes it difficult for him to move because of weakness. Breathing and coughing, needed at full post-operatively, may be rather ineffective.

Nutritional deficits can be corrected orally or parenterally, depending upon circumstances. So long as the patient can receive oral feeding, there is no need for parentral feeding. Natural or specially prepared oral feeding can correct malnutrition. When solid food cannot be taken it can be liquified.

In pyloric or duodenal obstruction, the liquid diet can be given through a small nasogastric tube. This is also the method if elderly patients resist feeding for psychological reasons. Aspiration is an everpresent hazard with tube feeding.

With complete obstructions, oesophogostomy, may be the suitable rout for feeding of patients.

To correct caloric deficit, the daily energy requirements are calculated within the normal range of 2000-3000 cal/day

Ex :	Protein	75 gm yield	300 calories
	Fat	150 gm »	1350 »
	Carbohydrate	200 gm »	800 »
			2450 »

Other losses as in ulcerative colitis or severe burns should be added to daily requirements. With encouragement, we may succeed to give patients double their requirements to prepare them for surgery in shorter time. This is referred to as oral hyperalimentation.

For parenteral feeding, convenient and safe solutions for intravenous administration led to the successful use of special solutions for the treatment of dehydration. Dextrose solutions are useful to supply water and small amounts of carbohydrate to spare protein. Hyperosmotic solutions as 25-50% glucose enable patients to get more calories in less volumes according to their physical status. Thus, giving more calories than normal may be possible intravenously without circulatory overload. This is referred to as parenteral hyperalimentation. Needless to say that other supplements can be added if possible and if useful such as vitamines.

For psychological preparation, the mental attitude of the patient towards surgery requires a consideration equal to his physical condition. The patient's experience of being a candidate of surgery, an odd happening in his life, is expected to disturb

his psychological condition irrespective of an apparent stable personality. A pre-operative visit from the anaesthetist to get the patient hope and confidence can induce a degree of mental relaxation, superior to the effect of preoperative sedatives. The anaesthetist should ensure that the patient of cold surgery should get a calm night sleep before the operation and the patient of an urgent surgery should pass to the theatre well sedated. The clever anaesthetist should succeed to establish a personal communication with his patient that may be reflected as a least disturbance of some important vital functions at his enterance to the surgical theatre.

Finally we stress the fact that the preparation of the surgical patient aims to enable him to meet anaesthetic and operative stresses. Preparation of a patient undergoing an urgent surgery needs a doctor with a good understanding background and a high degree of intelligence.

PRE-ANAESTHETIC MEDICATION

It is the duty of the anaesthetist to prescribe pre-anaesthetic medicaments to his patient Premedication is usually achieved by a combination of two pharmacologically different groups of drugs. The first is the parasympatholytic group and the second is the narcotic, sedative or tranquillizing group of drugs.

Parasympatholytic drugs used are atropine or hyoscine. They are given mainly to prevent serious cardiac arrhythmias mediated by the vagal nerves and to dry up secretions of the bronchial and salivary glands to prevent chest complications and to facilitate oral surgery. The secretions of the gut are also decreased which may prevent vomiting during and after anaesthesia in addition to being an aid in abdominal surgery, specially that the movements of the gut are inhibited. Many believe that hyoscine is a better drying agent than atropine. Parasympathalytics inhibit sweat secretion leading to diminished heat loss from the body, a point which should be considered to avoid hyperthermia in infants and children. The anaesthetist cannot depend on the pupil size as a critereon of the anaesthetic depth as parasympatholytic drugs dilate the pupils. The effects of atropine and hyoscine on the central nervous system are different. Atropine causes stimulation of the motor centres of the brain, which can antagonize the depressant effect of narcotics given simultaneously for premedication. Hyoscine is a cerebral depressant with an amnesic effect. It may be thought that hyoscine given for premedication can realise both effects needed from

a parasympatholytic and a sedative. It is better to avoid hyoscine administration in extremes of age. Hyoscine is preferred for premedication in patients with thyrotoxicosis due to the central depressant and less tachycardic effects.

Atropine is given in a dose of 0.6—1 mg and in young children in a dose of 0.1 mg per year. Hyoscine is given in a dose of 0.4—0.6 mg for adults.

Depression of the activity of the central nervous system is usually achieved by a dose of morphine. Morphine depresses the higher centres of the brain producing an inclination to sleep and uncaring attitude towards the approaching operation. It relieves pain and lowers the basal metabolic rate pooling at a decreased oxygen consumption. It causes slowing of the pulse rate and constriction of the pupils, both actions being antagonized by atropine or hyoscine. The main disadvantages of morphine are depression of the respiratory centre, depression of the cough centre, stimulation of the vomiting centre and the liability to histamine release. While morphine is indicated to a morphine addict patient to avoid the expected post-operative withdrawal symptoms, it is contraindicated in an asthmatic patient to avoid an acute asthmatic attack. In patients intolerant of morphine and those who are known to vomit severely after surgery, morphine may be replaced by pethidine or one of the phenothiazine type of drugs. Premedication producing sleep is usually referred to as basal narcosis. Heavy sedation is rarely advisable to avoid delayed post-anaesthetic recovery and to prevent inadevertant respiratory depression. Morphine is given in a dose of 10-15 mg for fit adults. Half the

dose is needed in old or debilitated patients. Although it is better avoided in children below five years of age, it can be given in consideration to body weight as 0.25 mg/kg. If the anaesthetist is ever in doubt over the correct premedication for an ill-person, he is advised to avoid morphine. An overdose of morphine or an inadvertant respiratory depression caused by morphine can be counteracted by nalorphan in a dose of 10—20 mg. Pethidine dose is 50—100 mg putting in consideration that 100 mg of pethidine is nearly equipotent to 15 mg of morphine.

The phenothiazines belong to the tranquillizer group of drugs. They are thought to act on the reticular formation of the brain. They have an anti-adrenergic effect causing vasodilatation and a fall in blood pressure with some protection of the body against shock. They have an anti-fibrillatory action with some drying effect on secretions. They have no analgesic effect and they are recently thought to have antanalgesic power, but they potentiate the action of other analgesics. They have specific anti-emetic effect which may be of value in prevention or treatment of post-operative vomiting. In some instances the hypotension caused by phenothiazines is deliberately induced to reduce operative bleeding. Anaesthetists may use these drugs to facilitate heat loss through widespread vasodilatation in the technique of induced hypothermia. Phenothiazines are known to have anti-histaminic properties which may be needed by the anaesthetist. Chlorpromazine (Largactil), promethazine (Phenergan) and promazine (Sparine) are the most commonly used members of this group of drugs. A dose of 25—50

mg given intramuscularly half an hour before operation is suitable to a fit adult patient.

Diazepam is sometimes given for premedication. Ten mg diazepam are equipotent to 10 mg of morphine regarding the soporific action but with less toxic effects. The experienced anaesthetist may find it suitable to use this drug in a particular patient for premedication.

Lastly, to get the optimum benefit of premedication, timing of its administration is of importance. It should not be given too early or too late by the intramuscular rout. Intravenous administration may be suitable in emergencies to get proper timed effects of drugs.

GENERAL ANAESTHESIA

STAGES AND SIGNS OF GENERAL ANAESTHESIA

The four pillars of proper general anaesthesia are the production of narcosis, the prevention of sensibility to pain, the disappearance of reflex responses to surgery and the provision of a sufficient degree of muscle relaxation needed for surgical interference.

It is customary to divide the progress of anaesthesia into four stages. But the third stage termed the stage of surgical anaesthesia, provides the four pillars of general anaesthesia allowing adequate levels for surgical interference. This stage is subdivided into planes which simply allow light or deep levels of surgical anaesthesia. While operations on the extremities need light levels, intra-abdominal operations need deep levels of surgical anaesthesia. It is the duty of the anaesthetist to tide the patient over the first and second stages of anaesthesia, to maintain him under the light or deep planes of the surgical stage of anaesthesia and to protect him from getting into a fourth stage of anaesthesia representing an anaesthetic overdose.

The sages of general anaesthesia are :

Stage I : Stage of analgesia.

Pain sensation is lost but consciousness is retained.

Stage II : Stage of excitement :

Consciousness is lost due to depression of higher brain centres. This allows subconscious manifestations and an over-reaction to all

forms of stimulation as noise and touch, leading to shouting, breath-holding or fighting and struggling.

Stage III : Stage of surgical anaesthesia :

This stage can be subdivided into planes or simply into light and deep levels.

Stage IV : Stage of overdose :

If the patient gets into this stage, he will be exposed to failure of respiration followed by failure of circulation and death.

This distinction of stages was originally described in relation to open ether anaesthesia without any premedication and cannot be strictly used to adapt the balanced anaesthetic techniques used nowadays. The anaesthetist should therefore stick to a classification based on clinical assessment of anaesthetic depth. He should not allow the surgeon to put his knife on the patient unless he is sure that the patient has reached the level of anaesthesia which provides the four essential pillars of surgical anaesthesia. He should then provide light or deep levels according to the requirements of surgery. He should finally prevent an overdose of the anaesthetic drugs used.

Signs observed to identify the proper anaesthetic depth are very numerous. But to simplify matters, the anaesthetist can get sufficient informations from three sites of observation, the repiration, the eyes and the response of the patient to surgical stimulation.

During the two stages before the surgical stage of anaesthesia, the patient's respiration may become irregular (due to breath-

holding) or looks faster than normal (tachypnoea) or deeper than normal (hyperventilation). At the entry of the required stage of surgical anaesthesia, respiration becomes completely regular. This is called the automatic respiration which is characteristic to the surgical anaesthesia. It remains to be decided whether this surgical anaesthesia is light or deep. If the patient's chest is moving, surgical anaesthesia is light. If the chest is not moving, the respiration is diaphragmatic as shown by the movement of the abdomen with each respiration and the surgical anaesthesia is deep. The anaesthetist has to guard against the stage of overdose where respiration shows progressive decrease in volume then jerky irregularities and unless anaesthesia is lightened respiration will cease.

Observation of the eyes yields informations related to the eyelash, the conjunctiva and cornea together with the pupil position and size.

The eyelash reflex denotes the reflex winking of the eye in response to a gentle touch of the eyelashes. So long as this reflex is present, the surgical stage of anaesthesia is not reached. The onset of the proper surgical anaesthesia is known by the loss of eyelash reflex.

The corneal and conjunctival reflexes are maintained until moderately deep anaesthesia. The anaesthetist is asked not to test these reflexes to avoid damage to these structures.

The pupils may be seen moving in the palpebral fissure. They may be seen fixed in a non-central position in the fissure. Both these positions of the pupil denote that the surgical stage of anaesthesia has not been reached when the pupils take a central position

in the palpebral fissure, then the proper surgical stage of anaesthesia has started. The pupils remain in this position till the end of anaesthesia.

Regarding the size of the pupils, they are observed to be normal or semidilated and reactive to light before reaching surgical anaesthesia. At the onset of surgical anaesthesia, they become constricted in light surgical anaesthesia, and progressively dilate as the surgical anaesthesia deepens being still reactive to light. An overdose of the anaesthetic usually produces dilatation of the pupils with absence of response to light due to the paralysis of the third nerve nucleus. These observations were reported by Guedel when he first described the stages of ether anaesthesia without premedication. Nowadays premedication usually includes atropine which dilates the pupils and morphine which constricts them. For this reason, the anaesthetist should not get solid informations from the pupil size to diagnose the anaesthetic depth.

Observing the patient response to surgical stimuli gives useful informations to diagnose anaesthetic depth. During light anaesthesia, a skin incision is accompanied by quick respiration, rapid pulse rate, a rise in arterial blood pressure, an increased tone of striated muscles, some lacrimation, reflex movement or laryngeal spasm. In this situation, anaesthesia should be deepened. During deep anaesthesia due to overdose, respiration becomes irregular, shallow or absent, pupils become dilated and irreactive to light, blood pressure decreases, pulse rate becomes too rapid or too slow and muscles become too flaccid. In this situation, anaesthesia should be lightened.

DRUGS USED FOR GENERAL ANAESTHESIA

Drugs used for general anaesthesia include parenteral and inhalational anaesthetics together with depolarizing and non-depolarizing muscle relaxants .

Parenteral anaesthetics include :

- Barbiturates :
  - Ultrashort acting barbiturates = i.e. Thiopentone Sodium
- Nonbarbiturates :
  - Eugenol derivatives = i.e. Propanidid
  - Steroid anaesthetics = i.e. Althesin, Minaxolone
  - Dissociative anaesthetics = i.e. Ketamine
  - Neurolept-anaesthesia = i.e. Fentanyl-Dehydrobenz peridol
  - Imidazol-carboxylate derivatives = i.e. Etomidate

Inhalational anaesthetics include :

- Gases :
  - Nitrous Oxode
  - Cyclopropane
- Vapours of liquids :
  - Ethers :
    - a) Non-halogenated = i.e. Di-ethyl ether
    - b) Halogenated = i.e. Methoxyflurane, Enflurane,
  - Halogenated hydrocarbons : = Fluroxene Halothane Trichlorethylene

Muscle relaxants include :

Depolarizing relaxants : Succinyl-choline

Non-depolarizing relaxants :

i.e. D-tubocurarine
Gallamine triethiodide
Pancuronium
Alcuronium
Fazadinium

This book will consider the agents with which the anaesthetist will be familiar in our country.

Parenteral Anaesthetics

The main drawback of intravenous anaesthetics is that a dose, once introduced into the circulation, cannot be recovered. An overdose is easy to be given if calculation of the dose is not accurate specially in young, old or debilitated patients. The wise use of a correctly designed dose is to give it slowly. As the normal arm to brain circulation time is between 10 and 15 seconds, intravenous anaesthetics are given over 30 seconds with the fingers palbating the pulse and the eyes observing respiration. In this way, the depressant effects on the circulatory and respiratory systems can be avoided.

Whenever intravenous anaesthetics are used, the anaesthetist should have his means for inflating the lungs with oxygen or air. He should have available analeptics and resuscitation drugs. He should be able to aspirate secretions and to introduce an endotracheal tube under vision by a laryngoscope to maintain a patent airway in the proper time. It is a good policy to administer an

electrolyte through an infusion set even if the patient does not need fluids as the anaesthetist may need the patent vein in an awkward time.

One characteristic of intravenous anaesthesia, is that the patient passes from the first stage to the third stage of surgical anaesthesia rapidly. The second stage is so short that excitement or struggling does not occur.

Ultra-Short-Acting Barbiturates

These are drugs administered by the intravenous rout, either as sole anaesthetics for minor surgery or for induction of anaesthesia for long operations.

Thiopentone sodium is the most familiar member of this series but methohexitone is another member which enjoys some popularity in other countries.

Advantages include :

— Simplicity of use with minimum of apparatus.

— Pleasant induction of anaesthesia.

— Non-flammable and non-explosive.

— Post-anaesthetic vomiting is rare.

— Cheap.

Disadvantages include :

— Respiratory depression : with rapid injection of correct dose or slow administration of a large dose of thiopentone.

— Hypotension : With rapid injection or over dosage. The drug should be given slowly and in small doses specially in shock states or when cardiovascular system is diseased.

— Laryngeal spasm : May occur and should be rapidly treated.

— Poor analgesia : Thiopentone is sometimes described to have ant-analgesic properties. No surgery is allowed except when the patient is truely anaesthetized.

— Prolonged recovery time : related to the total dose given specially if fractionated due to its cummulative effect. The patient should be accompanied home although he is apparently conscious.

— An inadvertent injection of thiopentone sodium into an artery may lead to gangrene of the limb. The mere injection of a small volume of the solution creates intense burning pain in the arm and hand. Once diagnosed, the anaesthetist leaves the needle in the artery and injects 5 ml of 1% procaine. Alternatively, papaverine 60 mg in saline may be injected. A severe spasm in the limb circulation may require a sympathetic block. In some cases it may be necessary to heparinize the patient. It may be a must to postpone the operation.

As thiopentone sodium is an irritant drug, an extravenous injection may lead to cellulitis and abcess formation. Even, when given intravenously, phlebitis may occur and this explains why 2.5% solutions replaced 5% concentrations as when diluted drug is given slowly, minimal contact with the endothelial wall of the vessel leads to minimal if any irrition.

Uses :

Thiopentone sodium may be used :

1) As a sole anaesthetic :

Once the patient has lost consciousness as observed by automatic respiration and loss of eyelash reflex, the anaesthetist should pay attention to the respiration and pulse. The drug can be used for short procedures like incision of an abcess or for removal of a nail. To prolong the effect of anaesthesia, it is better to avoid repetition of drug administration. Inhalation of a mixture of nitrous oxide and oxygen (70%, 30%) through a face mask is preferred.

2) For induction of anaesthesia :

A dose is calculated on body weight basis. The usual induction dose is 5—7 mg/kg for fit patients. For debilitated patients, a sleeping dose is given for induction which means a slow administration of the drug till loss of eyelash reflex.

This will be followed by other sequences for anaesthetic maintenance in prolonged operations.

Dissociative Anaesthesia

Ketamine is an anaesthetic which can be administered intravenously as well as intramuscularly, an advantage with children. Respiratory obstruction is rare with ketamine as the airway is more or less maintained, because the drug induces hypertonia in the skeletal muscles including the masseters. This agent is peculiar for its ability to raise the arterial blood pressure, an advantage in

the hypotensive patient and a disadvantage in the hypertensive patient.

This anaesthetic produces a state of altered consciousness during which the patient is disconnected from his environment and so ketamine is termed a «dissociative» anaesthetic. The drug induces post-anaesthetic psycho-mimetic reactions in the form of dreams and hallucinations which occur mostly is adults.

The drug can be used as a sole anaesthetic with advantage for children. It can also be used for induction of anaesthesia. The drug is supplied in two concentrations of 1% and 10%. The I.V. dose is 2 mg/kg which gives anaesthesia for 5—15 minutes. The I.M. dose is 10 mg/kg which gives anaesthesia for 15—25 minutes. In both circumstances, prolongation of the effect needs addition of smaller doses.

Inhalational anaesthetic gases

Nitrous Oxide

Nitrous oxide, the laughing gas is a weak anaesthetic but a potent analgesic. It is compressed in blue cyliders as a liquid. Together with oxygen, it is used as the vehicle for anaesthetic vapours in prolonged anaesthesia. It can be used as the main anaesthetic for dentistry. It can supplement thiopentone for minor surgical procedures.

Advantages include :

— Safety : provided nitrous oxide is administered with an adequate supply of oxygen, it can be considered the safest anaesthetic known.

— Pleasant on inhalation and non irritant to the repiratory tract. It was previously designated as the laughing gas.

— It has a rapid onset of action and it can be rapidly eliminated with rapid recovery.

— Non flammable

— Post-anaesthetic sequale are rare.

Disadvantages include :

— A weak anaesthetic although a potent analgesic.

— When given with oxygen, it deprives the patient from complete oxygenation which may be very valuable in patients with an ischaemic heart disease or anaemia.

Uses : Nitrous oxide is used in different ways :

— As a sole anaesthetic :

Combined with oxygen or air, nitrous oxide is used for dentistry or inision of an abcess.

— Comined with thiopentone or halothane and oxygen, it gives excellent anaesthetic conditions for minor surgery as removal of an ingrowing toe nail circumcission or cervical dilatation and currettage.

3) For maintenance of anaesthesia :

Nitrous oxide is used to provide deep anaesthesia as a vehicle for carrying and supplementing stronger anaesthetic vapours to the patient. It is usually given together with oxygen as 6 : 2 or 6 : 3 litres/minute.

— Nitrous oxide and oxygen or air can be used for painless labour in obstetrics for cylinders are sometimes available as premixed equal concentrations of nitrous oxide and oxygen for that purpose or during dressings of a burnt patient.

Inhalational Anaesthetic Vapours

Di-ethyl ether

Diethyl ether is a liquid with a boiling point of around 35°C which provides a potent anaesthetic vapour useful for all types of surgery.

Advantages include :

— Safety with a wide margin and it is thus useful for junior anaesthetists during the start of their training.

— Although, it induces myocardial depression in the isolated experimental heart, it stimulates the suprarenal glands of the intact animal or human to secrete catecholamines which maintain the blood pressure.

— It provides a good degree of analgesia which persists in the post-operative period.

— It provides a good degree of relaxation of striated muscles needed during many types of operations.

— Cheap.

Disadvantages include .

— Ether has a disagreable taste and pungent odour which irritates the tracheo-bronchial tree.

— Ether is flammable and explosive and cannot be used in surgical procedures using diathermy.

— Frequent post-anaesthetic vomiting.

Halothane

Halothane is a halogenated hydrocarbon with a boiling point of 50.2°C.

Advantages include :

— Non irritant to the tracheo-bronchial tree and can induce a pleasant induction of anaesthesia.

— Induction of anaesthesia and recovery are rapid.

— Halothane reduces arterial blood pressure which may be of advantage in reducing blood loss.

— It is non-flammable.

Disadvantages include :

— Being a potent anaesthetic, halothane is effective in small concentrations. It's administration needs calculated dosage through a specially calibrated vaporizer (Fluotec) and needs experience to avoid dangerous overdosage which may be fatal through both circulatory and respiratory depression.

— It produces relaxation of the uterus causing post-partum haemorhage, so, it is not suitable in obstetrics.

— It sensitizes the myocardium to exogenous or endogenous adrenaline.

— It has no analgesic properties and may provide an antanalgesic effect.

— The advantage of rapid recovery may be accompanied by the disadvantage of early post-operative pain sensation.

— Anaesthesia with halothane is followed by shivering which increases the body oxygen consumption.

— Halothane is doubted to induce a sensitivity like reaction with some liver damage giving a picture similar to viral hepatitis. Based on this, a previous history of jaundice or a history of a reaction following a previous halothane administration contraindicates the use of halothane. It is generally advisable to avoid repeated halothane administrations to the same patient within a short period of time.

Uses :

Halothane can be administered in the following ways :

— Given with nitrous oxide and oxygen halothane provides suitable anaesthetic conditions for operations not requiring profound muscular relaxation.

— Halothane is useful to supplement drugs used for induced hypotension.

— It provides good conditions whenever diathermy is used during surgery as it is non flammable.

— It is preferred for patients with chronic bronchitis or bronchial asthma.

TRICHLORETHYLENE

It is a liquid which provides a potent anaesthetic vapour. Its boiling point is 89°C. It provides an excellent degree of analgesia. It is suitable for light and unsuitable for deep anaesthesia.

Advantages include :

— Trichlorethylene is pleasant to inhale.

— It provides excellent analgesia without loss of consciousness

— It is non-flammable.

Disadvantages include :

— The use of trichlorethylene needs experience and a calibrated vaporizer (Tritec) to avoid overdose.

— It may cause cardiac arrhythmias in many forms as tachycardia, extrasystoles and auricular fibrillation.

— It may induce tachypnoea i.e. shallow rapid respiration

leading to hypoxia and carbon dioxide retension. This may add to the hazard of arrhythmias.

— If used in a closed-circuit, decomposition of its vapour may induce nerve palsies.

—Recovery may be delayed due to its slow excretion from the body.

Uses :

— Trichlor-ethylene can be used to provide light anaesthesia for surface surgery. An overdose is diagnosed by incidence of tachypnoea.

— Because of its high analgesic property, trichlorethylene, has proved highly effective in obstetrics. The inhalers used deliver 0.5% concentration of trichlorethylene in air under all conditions. The inhalers are connected to the patient by a wide-bore corrugated tube, expiratory valve and a face-mask. It has an air-vent near the mask as a safeguard. Once the patient gets unconscious, her fingers relax their pressure on the vent and only air is breathed from the inhaler.

— It can be used for burn dressings.

MUSCLE RELAXANTS

Depolarizing relaxants :

Succinyl choline or suxamethonium acts at the myoneural junction by a depolarizing effect which continues from three to five minutes after a single dose. This is considered a short acting relaxant which gives ideal conditions for endotracheal intubation. After thiopentone induction, succinyl choline injection provides relaxation of striated muscles including muscles of respiration and so the lungs should be inflated artificially by oxygen, intubation done and ventilation continued until the effect of the relaxant fades away. Succinyl choline is destroyed by pseudocholinestrase enzyme normally present in the blood. During maintenance of anaesthesia by ether for exemple, a more degree of relaxation necessary for a particular step of the abdominal surgery can be provided by injecting a small dose of succinyl-choline and artificially inflating the lungs. The dose of succinyl-choline is one mg/kg body

weight. The injection is followed by characteristic generalized muscular fibrillations called fasciculations before onset of muscle relaxation. These fasciculations may explain the muscle pains from which the patient complains on the second or third day of the operation. The more rapid the injection rate of this relaxant, the more coarse the fasciculations and tiring the muscle pain it induces and the more rise of the intraocular and intragasteric pressures. So, succinyl choline is better avoided in operations of open eye or in glucoma. A slow injection is advised with full stomach to avoid vomiting before intubation. In rare cases, the action of succinyl-choline is prolonged due to low levels of pseudocholinestrase enzyme as in cases of nutritional deficiency. Sometimes the blood contains atypical enzyme due to genetic aetiology. Sometimes, repeated doses of succinyl-choline reach a level which changes the depolarization block to a non-depolarization block. This is the dual block. In all these situations of prolonged apnoea, the wise treatment is patience and good ventilation. The anaesthetist is advised not to administer different medications which may make more harm than good, but to ensure that efficient ventilation prevents hypoxia and hypercarbia and thus his patient will be always safe.

Non-depolarizing relaxants :

This group of drugs act by competitive inhibition with acetyl-choline at the myoneural junction. Their effect is longer than that of succinyl-choline and so they permit an upper abdominal operation to be done under light general anaesthesia. Instead of giving

deep ether or halothane anaesthesia for relaxation in abdominal surgery, we give light ether or halothane anaesthesia and a non depolarizing long acting muscle relaxant. Once the relaxant is given, the lungs should be artificially ventilated by continuously squeezing the reservoir bag. The dose of d-tubocurarine (curare) is 0.5 mg/kg and the duration is 30—45 minutes. The dose of gallamine triethiodide (Flaxedil) is 2 mg/kg and the effect persists for 20—30 minutes. These drugs may reduce the blood pressure due to histamine release and ganglion blockade. Recent drugs as pancuronium, alcuronium and fazadinium provide more stable cardiovascular system. There is great variations in responses of patients to these drugs according to different factors such as kidney function, electrolyte balance and nutritional state. So the use of these drugs need experience and a lot of scientific background.

Non-depolarizing muscle relaxants have an antidote. Anticholinesrases as neostigmine (prostigmine) can reverse the effect of relaxants when needed Neostigmine produces muscarinic effects such as bradycardia, stimulation of mucous secretions of the respiratory tract and contraction of the gut. These unwanted effects can be inhibited by administering atropine intravenously few minutes before the intravenous administration of neostigmine. The dose of neostigmine needed is up to 5 mg. Atropine is given in a dose of one mg for each 2.5 mg neostigmine. Neostigmine should be given slowly until muscle paralysis has been reversed completely depending on different factors as the amount of the muscle relaxant given, when the relaxant was given and the kidney function. The administration of neostigmine needs much experience and practice to avoid complications.

THE ANAESTHETIC MACHINE

For the administration of anaesthesia, an anaesthetic machine is used and it should be checked from time to time to ensure safety of the patient. This machine consists simply of :

— Cylinders of compressed gases : mostly oxygen and nitrous oxide.

— Reducing valves : through which the pressures of compressed gases are reduced to clinically useful levels.

— Flowmeters : which permit a desired flow of oxygen or oxygen and nitrous oxide mixture in terms of litres per minute needed by the patient

— Vaporizers : These are devices containing the liquid anaesthetic and which facilitate its vaporization.

— A reservoir bag : through which patients inhalations are taken. Together with a corrugated tube ending in the facepiece, it is called the Magill attachement — The face-piece fits the nose and mouth of the patient and is equipped with an expiratory valve for the release of each exhalation.

The machine including these constituents is the continuous flow semi-open apparatus which is the main anaesthetic equipment available in every surgical theatre in Egypt.

Cylinders :

The cylinders contain compressed gases under high pressures. Oxygen is compressed under a pressure of 120—132 atmospheres, while nitrous oxide is compressed under a pressure of 51 atmospheres. The walls of these cylinders should be built in a way to withstand high pressures. Oxygen pressure in the cylinder is about 2000 bound per square inch (140 kg/cm) while nitrous oxide pressure in the cylinder is about 750 bound per square inch (50 kg/cm), both at 15°C. Each cylinder is painted according to a standard code of colours :

Oxygen : Black, with white shoulders.

Nitrous oxide : Blue.

Carbon dioxide : Grey.

Cyclopropane : Orange

Reducing Valves :

The cylinders of compressed gases are equipped with reducing valves which are not interchangeable. A reducing valve consists of a spring loaded diaphragm to connteract the pressure inside the cylinder. A reducing valve for an oxygen cylinder always carries a pressure gauge indicating the amount of oxygen in the cylinder. When nitrous oxide gas is compressed in a cylinder it liquifies under a critical temperature and pressure. So long as a cylinder contains some nitrous oxide liquid, the vapour on the surface of this liquid will give a constant pressure at a constant temperature. A pressure gauge on the reducing valve would give no indication of the amount of nitrous oxide in the cylinder until the last drop

of the liquid turns into vapour. Thus, a reducing value for a nitrous oxide cylinder does not essentially include a pressure gauge. The only way to know the contents of a nitrous oxide cylinder is to find out the difference between the weight of the empty and full or partially full cylinder. The weight of an empty cylinder is thus always stamped on the neck of the cylinder.

Flowmeters

Flowmeters are arranged in a container according to the gases used ; oxygen, nitrous oxide, carbon dioxide and cyclopropane. Flowmeters lead into a comon channel where gases mix. Access of gases to flowmeters is by needle valves which are controlled by the anaesthetist.

There is a big variety of flowmeters, but the rotameter is the common type attached to anaesthetic machines owing to its high accuracy. It contains a bobbin which spins in the gas flow clear of the walls of a tapered glass tube to avoid friction. The bobbin can indicate the gas flow as graduated on the glass tube as litres per minute. From flowmeters, mixed gases pass through a metal tube to which various vaporizers may be attached.

Vaporizers :

The simplest vaporizer is the ether bottle. It consists of a sleeve valve which directs part of the gas flow into a glass bottle containing ether liquid. Ether vapour concentration will be affected by four factors, the rate of gas flow, the surface area of the liquid, the way in which the gas impinges on the liquid and the

temperature of the liquid. The temperature of the liquid is the only factor which is not under the control of the anaesthetist. As the ether bottle cools during use, its vapour pressure falls, and it becomes difficult to control the concentration of the delivered vapour.

The more potent anaesthetics should be accurately calibrated to deliver calculated concentrations. Thus more sophisticated improved vaporizers incorporating thermocontrol mechanisms were essential. They may be calibrated for specific anaesthetics as the Fluotec for halothane, the Tritec for trichlorethylene, the Pentec for methoxyflurane and the Ethertec for enflurane. Administration of halothane for example needs 2—4% for induction and 1—2% for the maintenance of anaesthesia.

The Magill Attachment

It consists of a rubber reservoir bag, about one meter of a wide bore corrugated rubber tubing and a face-piece with a spring loaded expiratory valve. It requires a gas flow equal to at least the minute volume of the patient respiration to prevent rebreathing and accumulation of carbon dioxide.

The anaesthetic machine is arranged round a trolley upon which supplementary pieces of apparatus are available like the laryngoscope and the mouth gag. All the rubber components of the machine are made of an antistatic material to discourage the formation of static discharge to guard against explosion when using inflammable anaesthetic vapours.

ENDOTRACHEAL ANAESTHESIA

The AIRWAY :

One of the most important principles of general anaesthesia is to keep a clear patent airway. It means that the anaesthetist should be sure that the upper respiratory airway is kept free from an obstruction or a possibility of its occurrence. This principle applies when general anaesthesia is administered through an intravenous or inhalational rout or through using a technique including both routes of administration. It also applies to patients who left the surgical theatre but did not yet return to full consciousness. It is also applicable to patients who are unconscious from any reason whatsoever.

When the airway is obstructed, the patient will suffer from oxygen lack (hypoxia leading to hypoxaemia) and carbon dioxide retension (accumulation leading to hypercarbia). The patient becomes exhausted due to his increased respiratory efforts. He will be suffocated with the possibility of losing life if active measures to release the obstruction and other sorts of treatment are not taken instantaneously. The dangers and the management will depend on whether the obstruction is partial or complete.

The signs of respiratory obstruction during anaesthesia are :

— Diminusion or absence of respiration

— Indrawing of muscles in intercostal spaces and suprasternal region

— The use of accessory muscles of respiration

— Cyanosis.

The most common causes of airway obstruction are :

— Falling back of the tongue

— Laryngeal spasm

— Accumulated secretions, blood or vomitus or foreign body in the mouth, pharynx or upper respiratory tract

— Mechanical fault as an empty oxygen cylinder, damaged reducing valve, leakage in the anaesthetic machine or a kinked endotracheal tube.

The management of respiratory obstruction depends on :

1) The correct management of the causes :
   - i) Extension of the neck and drawing the jaw foreward to seperate a falling tongue from the posterior pharyngeal wall
   - ii) Prevention of direct or reflex irritation of the larynx and administration of oxygen in cases of laryngeal spasm.
   - iii) Aspiration of accumulated secretions, blood or vomitus and removal of foreign bodies causing the obstruction.
   - iv) Proper correction of any underlying mechanical fault.

2) Ending the anaesthetic administration and giving enough oxygen.

3) Performing artificial respiration until normal breathing is established.

4) In an unconscious patient or in presence of cyanosis, an endotracheal tube may be passed easily to continue administration of oxygen specially if signs of hypoxia are severe or respiratory depression is apparent or circulatory failure is completing the picture where circulatory support must be simultaneously carried out.

The endotracheal tube :

Many a time, the anaesthetist feels a great need to guarantee a patent airway of his patient during anaesthesia. This is established by using an endotracheal tube. It enables him to practice artificial ventilation and at the same time not to inflate the patient's stomach as the lungs are inflated. The use of muscle relaxants particularly the short acting suxamethonium has introduced ideal conditions for easy endotracheal intubation.

The indications for endo-tracheal intubation are :

— Operations on head and neck, including the ear, nose and throat and dental surgery.

— Abnormal positions of the patient as the prone or steep Trendelenberg one.

— Upper abdominal surgery needing artificial respiration due to induced muscle relaxation.

— Emergency operations with full stomach to protect the lower respiratory tract from the danger of inhalation.

— When pathological conditions necessitate intubation as the need for continuous aspiration of secretions in bronchiactasis.

— For management of some patients in the intensive care unit for some days on artificial ventilation. This is either post-operatively or due to poleomyelitis, tetanus or myasthenia gravis.

The endotracheal tube may be fitted with an inflatable cuff. This cuff, when inflated, can form an airtight joint between the tube and the respiratory passages and in this way it gives perfect conditions for artificial respiration (intermittent positive pressure ventilation). The cuff also renders a most valuable service in preventing material such as saliva blood or vomitus from gravitating into the lower respiratory tract.

TRACHEOSTOMY

When artificial ventilation of the patient becomes necessary for more man several days, tracheostomy has to be performed and a cuffed tube inserted. In order to facilitate the changing of the tube, the operation should not be done at too low a level in the neck, and where possible a flap of tracheal wall should be stitched to the skin edge.

A tracheostomy tube is the safest method of connecting the patient needing prolonged artificial ventilation to the ventilator. Its use is, however, by no means free from risk. For exemple if the tracheostomy tube is improperly inserted in the tissues of the neck only, subcutaneous emphysema may result. When a tube is pressing on the backwall of the trachea, ulcerations and airway obstruction may occur. Other hazards may be related to the tube cuff as cuff herniation or over-inflation.

ADMINISTRATION OF GENERAL ANAESTHETICS

The rôle of the anaesthetist administering general anaesthesia may be compared to the rôle of the pilot leading his plane during a journey. The experienced pilot makes a smooth take off, an uneventful flying and an easy safe landing of his plane. In a similar way, the trained anaesthetist, makes a smooth induction, an uneventful maintenance and an easy emergence from the anaesthetic effect. The job of both the pilot and the anaesthetist is happily fulfilled through their meticulous attention and intelligent guidance whatever the technique they adopt during the journey.

ADMINISTRATION OF INHALATIONAL ANAESTHETICS

Inhalational anaesthetics can be administered by one of four methods :

I — Open method

II — Semi-open method

III — Semi-closed method

IV — Closed method

I — Open method :

The liquid anaesthetic is dropped on a gauze surface covering a mask applied to the face (Schimmelbusch mask). In this way the anaesthetic is vaporized. The vapour is then drawn into the

lungs mixed with atmospheric air which is inhaled through the mask.

Administration should begin slowly until the patient becomes adapted to the vapour without breath holding then the dropping rate can be gradually increased until the surgical stage of anaesthesia is reached.

It may be sometimes helpful during induction of anaesthesia by the open method to allow a slow trickle of oxygen under the margin of the mask.

In the difficult environment, anaesthesia can be maintained by the open drop method. A Guedel's airway helps to support the tongue and facilitate aspiration of secretions from the pharynx

II — Semi-open method :

It simulates the same technique used for open anaesthesia. But when the mask is covered with gamgee gauze and its sides are built to make tight connection with the face, carbon dioxide will be accumulated from the patients exhalations. The aim of the semi-open method is to increase the depth of respiration to hasten induction of anaesthesia. This occurs through the inspired carbon dioxide air mixture.

The open and semi-open methods are simple and safe but unpleasant to the patient. They are replaced by the more advanced methods of induction and maintenance of general anaethesia.

III — Semi-closed method :

A fresh gas inflow is continuously introduced from the gas cylinders into the anaesthetic machine. The patient inspires from the reservoir bag of Magill's attachement. On exhalation, excess gases escape to the atmosphere through an expiratory valve. The system is called non-rebreathing if all the exhaled gases escape through the expiratory valve. It is called a partial rebreathing system if only a part of the exhaled gases escape through the expiratory valve.

IV — Closed method :

In this method, enough oxygen is delivered to supply the patient's needs per minute. In order that this oxygen can be inhaled again and again in a closed system, exhaled carbon dioxide produced by metabolism must be removed by an absorbent ; the soda lime. So, the closed method involves complete rebreathing and no access to the atmosphere either on expiration or on inspiration. For practical purposes, there are two modifications of the closed method. These are the to-and-fro and the circle methods. Certain inhalational anaesthetics cannot be administered in a closed system because of decomposition. Trichlorethylene decomposes due to its reaction with the soda lime used for carbon dioxide absorption as toxic products produced may induce toxic nervous manifestations to the patient.

Ayre T-tube :

The technique using the Ayre T-tube is usually used for anaesthesia in children. The expiratory arm has a rubber extension

which represents the reservoir bag. Rebreathing is prevented by controlling the length of the expiratory tube in relation to the volume of fresh anaesthetic gases and the child minute volume. If the expiratory tube is very short, the child, during inspiration, will inhale anaesthetic gases and oxygen from the delivery tube which will be much diluted by inhaling room air at the same time through the short expiratory tube. In this way, the system is considered as an open one. On the other hand, if the expiratory limb is too long, rebreathing from this limb will occur at the time of inspiration. A small bag with an open end can be attached to the end of the expiratory limb. It acts as an indicator for inspiration and facilitates its controlling by the anaesthetist. This is termed the Ree's modification of the Ayre-T-tube.

ADMINISTRATION OF INTRAVENOUS ANAESTHETICS

Intravenous anaesthetics are administered for different purposes :

(1) Induction of anaesthesia : In long operations, anaesthesia is started by an intravenous agent so that the patient passes rapidly to the surgical stage of anaesthesia, thus, bypassing the second stage of excitement and struggling. Maintenance of anaesthesia is achieved through the use of other agents.

For induction of anaesthesia, only calculated doses are given to the patient. Thiopentone is given in a dose of 5—7 mg/kg body weight and ketamine in a dose of 2 mg/kg body weight.

(2) As a sole anaesthetic : In short operations, surgery may be carried out under nearly the same doses of intravenous anaesthetics used for induction of anaesthesia. Anaesthesia may be supplemented with nitrous oxide and oxygen, fluothane and oxygen or nitrous oxide, fluothane and oxygen through a face-mask to facilitate or prolong surgery.

Ketamine as a sole anaesthetic may be given intramuscularly in a mean dose of 10 mg/kg body weight to facilitate short surgical procedures. Addition of nitrous oxide and oxygen or repetition of smaller ketamine doses may facilitate or prolong surgery.

(3) Total intravenous anaesthesia :

Recently, the problem of pollution of surgical theatres by traces of anaesthetic gases and vapours of liquids has become acute. Pollution was found to threaten health of theatre personnel leading to bone marrow depression, miscarrages, congenital malformations of their newborns and possibly malignancies. The only long term solution of this problem appears to develop a safe, effective intravenous anaesthetic technique. At the present time, no intravenous anaesthetic drug possesses all the characteristics of an ideal agent. So, no intravenous anaesthetic can be used as the sole agent in long surgical procedures due to the toxicity of the large doses needed. To give an intravenous agent for long operations, seperata drugs should be used to provide hypnosis, muscle relaxation and suppression of the reflex responses to noxious stimuli. This balanced anaesthetic technique including small total doses of an intravenous anaesthetic in addition to other drugs to provide relaxation and or analgesia is the basis of the new technique termed intravenous total anaesthesia. Intravenous anaesthetics are given during surgery either intermittently or by a drip through an intravenous infusion.

ADMINISTRATION OF GENERAL ANAESTHESIA IN CLINICAL PRACTICE

The technique of balanced anaesthesia is the basis of the clinical use of general anaesthesia. To avoid harmful effects of large doses of one single drug, smaller doses of few drugs can give the essential requirements of general anaesthesia, namely, hypnosis, analgesia, muscle relaxation and reflex suppression. The grouping of the different agents used form a countless number of balanced anaesthetic techniques depending on the choice and experience of the particular anaesthetist. Some of these techniques can be mentioned as exemples (after patients are premedicated) :

(I) For short operations :

i — An intravenous anaesthetic alone.

ii — An intramuscular anaesthetic alone.

iii — An intravenous anaesthetic supplemented with an inhalational agent and oxygen through a facemask and an anaesthetic machine.

iv — An inhalational agent and oxygen using a facemask and an anaesthetic machine.

(II) For long operations

(A) On the head and neck, extremeties or lower abdomen (spontaneous respiration)

i — For induction of anaesthesia :
An intravenous anaesthetic or a potent inhalational anaesthetic

ii — For maintenance of anaesthesia :

An inhalational anaesthetic and oxygen.

iii — If an endotracheal tube is needed, it can be put by using a short-acting muscle relaxant.

(B) On upper abdomen or chest (controlled respiration)

i — For induction of anaesthesia :

An intravenous or a potent inhalational anaesthetic

ii — Tracheal intubation using suxamethonium.

iii — For maintenance of anaesthesia :

a) Nitrous oxide and oxygen

b) An inhalational anaesthetic and oxygen and may be nitrous oxide. Long relaxation is achieved by a non-depolarizing muscle relaxant whose effects are antagonized at the end of operation by an anticholinestrase and atropine.

c) Anaesthesia may be maintained using the technique of total intravenous anaesthesia. It entails giving the intravenous anaesthetic i.e. Ketamine, Althesin or Etomidate, by infusion either intermittently or by drip during maintenance. Muscle relaxation is achieved by using a non-depolarizing muscle relaxant whose effects should be reversed at the end of anaesthesia. As relaxants are used, artificial ventilation should be carried out through an endotracheal tube during the whole anaesthetic duration.

COMPLICATIONS OF GENERAL ANAESTHESIA

Complications of general anaesthesia are countless because the aetiological factors are very variable. These belong to the hospital, the theatre itself, the anaesthetist, the patient and the surgeon Prophylaxis is the best policy to minimize complications. But prophylaxis never returns to the anaesthetist alone.

The complications can be categorized in the following way :

I. cardiac and circulatory :
   1) Shock
   2) Cardiac arrest
   3) Post-operative venous thrombosis
   4) Pulmonary embolism.

II. Pulmonary :
   1) Bronchitis
   2) Lung collapse
   3) Pneumonia
   4) Lung abcess
   5) Pulmonary oedema
   6) Cross infection

III. Central nervous system :
   1) Hypoxia and its sequalae
   2) Peripheral nerve palsies

IV. Renal :
   From acute tubular necrosis to renal failure

V. Hepatic :
   From a hepatitis like reaction to terminal hepato-renal failure.

VI. Ophthalmic :

1) Irritant effect of vapours or sterilizing solutions.
2) Thrombosis of the central artery of the retina.
3) Acute glucoma may be enhanced by drugs causing pupillary dilatation.

VII. Fire and explosion :

The underlying factors are : an explosive mixture of oxygen and flammable substance together with an ignition source.

VIII. Electrical :

1) Cardiac arrest.
2) Burns.
3) Neurological damage.
4) Explosions.

IX. Chemical :

Pollution of anaesthetic theatres by anaesthetic gases or vapours above the minimum acceptable level has been blamed, on chronic inhalation by theatre personnel, to cause :

1) Miscarriages
2) Still-births
3) Pre-mature births.
4) Liver damage
5) Performance decrements
6) Fatigue
7) Headache
8) Malignancy

Inhibition of immune responses of theatre personnel may be a prominent factor.

POSITIONING OF PATIENTS

Patients lie on the surgical table in different positions to facilitate an easy access to specific surgical procedures. At the same time these positions should not impose unwarranted hazards on the vital functions of patients as these positions may be maintained for prolonged periods during anaesthesia. They should induce minimal interference with the circulation and respiration. Positioning should provide good support of the patient on the table. There should be adequate padding to areas of contact with the table to prevent nerve injuries. Adequate measures may prevent complications. Intermittent pumping or massaging of the gastrocnemius muscles for exemple may decrease the incidence of postoperative deep venous thrombosis.

The most common position is the supine or dorsal recumbent position which embrasses the patient the least. In the prone position, care should be taken to maintain a free airway, to support the chest and to avoid obstruction of venous return to the heart. In Trendelenberg position the table is tilted to about 15 degrees and well padded shoulder braces are attached to fix the patient. In the kidney position, the patient is turned on his side and the lower leg is flexed to 90 degree angle while the upper leg is kept straight. While raising the bridge of the table, care is taken to avoid obstruction of abdominal vessels. In the lithotomy position, popliteal knee supports should be well padded to prevent thrombosis of superficial vessels.

To test the position of the patient, the anaesthetist can estimate whether or not a particular position can be tolerated in the conscious state. It should be comfortable even if maintained, and not causing any physical injury or physiological insult.

LOCAL ANALGESIA

Sometimes the anaesthetist is confronted with an operation which urges him to choose a local technique. This happens usually when surgery is minor. In such conditions, a patient with a full stomach is a good subject for local analgesia, on condition that the patient should be co-operative. Local analgesia is a justified choice when the life of the patient is endangered by being unconscious. Sometimes the nature of the operation is the deciding factor i.e. drainage of an empyema or repair of a broncho-pleural fistula. When the cooperation of the patient is needed during the operation i.e. internal ear operations, local analgesia is preferred. In all conditions, sepsis near the field of injection will be a contraindication as local anaesthetics do not act in the acidic medium of pus.

As a rule, premedication for patients undergoing surgery under local analgesia should be heavier than for general anaesthesia.

Various drugs are used for local analgesia. These include : Lignocaine (Xylocaine), Prilocaine (Citanest), Procaine (Novocaine) Cocaine, Bupivacaine (Marcaine), Mepivacaine (Carbocaine), Cinchocaine (Nupercaine) and Etidocaine (Duranest)

All local analgesics, except, cocaine, cause vasodilatation. Adrenaline may be added to counteract this effect, to prolong the analgesic effect and to reduce the chances of a toxic dose reaching

the circulation. The usual concentration of adrenaline is 1/200.000 and the total dose should not exceed 0.5 mg. However, adrenaline should not be added to local analgesics injected at the root of the penis or roots of fingers in ring anaesthesia to guard against gangrene of these organs.

Techniques of local analgesia include :

— Surface analgesia.

— Infiltration analgesia.

— Nerve block

— Field block

— I.V. regional analgesia for extremities

For surface analgesia cocaine 1-4% is always used as a spray, ointment or cream. A lozenge of amethocaine (60 mg) may be sucked before laryngoscopy or bronchoscopy. Surface analgesia is useful for submucous resection and nasal polypi, incision of quinsy, minor eye surgery and cystoscopy.

For infiltration analgesia 0.5% lignocaine with adrenaline is useful. The technique is easy and requires no anatomical knowledge and is effective. It may be combined with general anaesthesia to provide a bloodless field by making use of the vaso constrictor and to maintain a light anaesthetic plane.

Nerve block requires an exact knowledge of the anatomy, for success. Lignocaine 1—2% is usually used. For ring anaesthesia, the analgesic solution infiltrated around the base of the finger

affects the digital nerves. In this situation neither adrenaline nor a tourniquet is used. For extraction of the molar tooth, the inferior dental and lingual nerves are blocked in the region of the mandibular foramen. The intercostal nerves can be blocked below their respective ribs for relief of pain of fractured ribs or pain after upper abdominal surgery. Care should be taken to avoid damaging the underlying pleura and lungs causing pneumothorax.

For field block, a frame of local analgesic solution is infiltrated across the path of the nerves supplying the surgical field if the anatomy of the operation permits.

Intravenous local analgesics may be injected in the extremeties for minor operations. The limb is exsanguinated and a tourniquet applied over the upper arm and inflated to above systolic B.P. before injection. Prilocaine 0.5% is injected through a pre-cannulated vein on the dorsum of the hand, in volumes of 40 ml for an arm and 80 ml for a leg. The method is always easier in the arm with a small bulk of muscles. Analgesia develops within 10 minutes. Release of the tourniquet should be gradual after 20 minutes minimum from injection for fear of toxic reactions. All operations on the upper limb specially those needing the patient cooperation as tendon repair can be done under intravenous local analgesia. A patient with a full stomach is an indication Local analgesia is not without danger and idiosyncrasy and overdose are the main sources of complications.

Immediately following injection of small quantities of local analgesics, sudden collapse sometimes occur. But it is doubted if

this is true idiosyncrasy. In many instances, it may be due to inadvertent intravenous injection of the drug. In such a happening, the anaesthetist should tilt down the head of the table, give oxygen, start an infusion and give a vasopressor agent.

It should be planned by the anaesthetist to avoid the maximum dose of the local analgesic he gives to minimize its risks. The maximum dose for Cinchocaine, Bupivacaine and Cocaine is 150 mg, for Lignocaine is 500 mg, for Prilocaine is 600 mg and for Procaine is 1000 mg. All local analgesics act for durations of 1—2 hours except cocaine which acts for around 20 minutes and Bupivacaine which acts for 4—5 hours.

It should be noted that the site of injection is also important. A relatively small amount of local analgesic injected in a highly vascular area is more dangerous than a larger dose where the blood supply is limited.

Overdosage of local analgesics is manifested by circulatory and cerebral signs. Bradycardia and hypotension signify depression of the myocardium and blocking of the conducting mechanism of the heart. Convulsions preceded or followed by cerebral depression indicate cerebral affection. Management entails, checking the patient airway, administering oxygen, performing artificial respiration if necessary after injecting a small dose of suxamethonium and passing an endotracheal tube. This also can control convulsions safely. Vasopressors can support the circulation. If cardiac arrest occurs adequate measures are taken.

In some situations, adrenaline contained in the analgesic solution may cause a reaction. The patient is pale and his pulse is very rapid, nearly impalpable and irregular. The blood pressure shows a brief dangerous rise. If the patient is conscious, he looks anxious and complains of headache, nausea and palpitations. Many cases end in ventricular fibrillation and death. The systemic effect of adrenaline is transient but some patients do not withstand such a reaction while others recover spontaneously.

SPINAL ANALGESIA

Spinal analgesia plays an important rôle in anaesthetic field in our country up till the present time. It is a simple technique which provides a cheap type of analgesia. It induces an excellent degree of muscle relaxation which satisfies the requirements of surgical interference. It causes some hypotension which limits the amount of lost blood during surgery. The lungs are not irritated and post-operative chest complications are minimal. Some patients are happy to keep conscious during the operation. Spinal analgesia prevents the fire and explosion hazards. It helps in the solution of the problem of pollution of operative theatres by anaesthetic gases and liquids with its harmful effects on health of theatre personnel.

On the other hand spinal analgesia is not without risks. The higher the level of spinal analgesia, the greater the fall in arterial blood pressure. This is related to the degree of paralysis of the sympathetic nervous system. Hypotension may have its own risks and there is the probability that bleeding may occur when the blood pressure rises again at the end of surgery. It is important to notice that vomiting occuring during spinal analgesia is always associated with hypotension and that this denotes the early onset of cerebral anaemia. This should be managed instantaneously by administration of vasopressors and oxygen after clearing the airway. Spinal analgesia is peculiar for the onset of high incidence of post-spinal headache. Concerning its aetiology, the consensus is that it results from leakage of C.S.F. through the dural puncture site, at a rate greater than its production by the choroid plexus. The brain loses its fluid cushion and has a tendency to sag particularly in the upright position and tension is placed on pain sensitive anchoring structures Typical spinal headache is occipital, at

the vertex or frontal behind the eyes. It may be associated with nausea, vomiting or diziness and is exaggerated in the upright position. It usually appears on the first to the third post-spinal day and lasts from one day to one year, ranging in quality from mild to severe. Headache after spinal puncture not showing these characteristics is described as atypical. Tourtellotte et al (1964) listed alphabetically 49 methods of treating post-lumbar puncture headache. These included the use of fine spinal needles, keeping patients in recumbent position, abdominal binders, analgesics, sedatives and epidural saline injection. Recently, it was noted that when tapped C.S.F. is bloody, post-spinal headache is rare due to sealing of the puncture site by coagulated blood. Blood patch technique for treatment of typical post-spinal headache includes the epidural injection of autologous blood near the previous site of spinal injection. This technique was found to provide 98.4% success in pain relief by Ostheimer et al (1974).

Rare complications of spinal analgesia include meningitis and post-operative neurological sequelae as cauda equina lesions.

Spinal analgesia is useful for lower abdominal and lower limb operations. It is specially indicated in presence of chest infection or full stomach. Spinal analgesia is preferred in presence of hepatic or renal disease. On the whole, the use of spinal analgesia depends on the choice of the anaesthetist and the co-operation of the patient.

Spinal analgesia is contra-indicated in children and ill-feeble patients. It is not used in presence of skin infection, deformities of the vertebral column or in patients with central nervous system diseases. It is not suitable for patients with unsound cardiovascular system or patients with severe abdominal distension.

Drugs used for spinal analgesia are the same drugs used for local analgesia. The most widely used in our country is mepiva-

caine (carbocaine 4%) which gives an analgesic duration of 1—2 hours, or bupivacaine (marcaine) which provides analgesia up to 5 hours. These are heavy solutions with a specific gravity greater than that of C.S.F. and so will fall at injection. This fact should be considered when a head down position is required by the surgeon or to combat a fall in blood pressure. As drugs are fixed in about 10—15 minutes after injection, a head down tilt is better avoided in the first 15 minutes to avoid total spinal analgesia caused by cephalad spread of the drug.

Patients undergoing surgery under spinal analgesia need heavier pre-medication than those using general anaesthesia, in order to allay their anexiety. They need meticulous observation by the anaesthetist during the course of the operation with repeated checking of their general condition, their pulse and blood pressure. Sometimes, a minor tranquillizer is given I.V., as diazepam in a dose of 5—10 mg, to allay restlessness during the course of anaesthesia. The author sometimes gives ketamine in a dose of 1 mg/kg I.V. to provide dissociation of the patient from his surroundings. Ketamine (in such a small dose) supplementing spinal analgesia has the advantage of maintaining the arterial blood pressure without post-spinal hallucinations. After the operation, the anaesthetist should guard against headache. The patient is moved to a semi-dark calm room for few hours. He is asked to remain recumbent in a supine, lateral or prone position and not to raise the head which usually aggravates the headache. He should receive analgesics once he feels pain. He should be encouraged to drink as much as possible.

EPIDURAL ANALGESIA

Epidural analgesia entails the deposition of the local anaesthetic into the spinal cord but outside the dura. Synonymus terms are thus peridural or extradural analgesia. Analgesia is obtained by blocking spinal nerves in the epidural space as they emerge from the dura and then pass into the intervertebral foramen. A segmental block is produced chiefly of spinal and sensory nerve fibres. Motor fibres are partially blocked. Deposition of the local analgesic may be accomplished at the thoracic, lumbar or caudal areas, as the epidural space extends from the base of the skull to the sacral hiatus.

The factors controlling the extent of epidural analgesia include the site of injection, the volume of solution, the speed of injection, the position of the patient and the specific gravity of the injected solution. In order to recognize the position of the needle in the epidural space, methods depend on taking the advantage of the negativity of that space or utilizing the sudden disappearance of resistance when the ligamentum flavum is penetrated. The technique is described as being difficult, but by training, more success is approached.

The drugs mostly used for epidural analgesia include Lignocaine (Xylocaine 2%), mepivacaine (carbocaine 1.5%) and bupivacaine (marcaine 0.5%). The author feels that epidural analgesia has a role to play whenever general or spinal anaesthesia seems to be contra-indicated. It is of value in poor risk patients, with cardiac

or pulmonary disease and in the presence of metabolic disturbances. In addition, it has a definite role in obstetric analgesia and in management of chronic pain.

But, it is wise to abandon the technique in uncooperative patients, shock states, coagulation defects, previous laminectomy or in presence of local skin inflammation.

The anaesthetist should put in his mind a guide to the site of injection and the volume of local anaesthetic to be injected to get different blocks. For perineal operations 10—12 mls are injected at L_3 — L_4. For operations on the extremities, 12—14 mls are injected at L_2 —L_3. For Lower abdominal surgery 14—16 mls are deposited at L_1 or L_2 interspace. For upper abdominal operations 16—18 mls are given at T_{12} or L_1 interspace.

Epidural analgesia may involve a single dose technique or a continuous technique in which repeated injections of the local anaesthetic can be given through a fixed catheter at the times needed. The last technique is valuable in obstetric analgesia or in management of chronic pain.

Epidural analgesia has some advantages over spinal analgesia. The incidence and magnitude of arterial hypotension is less. The risk of post-spinal headache and serious neurological sequelae is much less. Theoretically, it is safer to use a continuous technique in the epidural than the sub-arachnoid space. In contrast, epidural analgesia has certain disadvantages when compared with sub-arachnoid analgesia. The technique is more difficult and requires skill but still success is not definite. The onset of analgesia is slower and the extent of the block and its intensity are less certain.

SPECIAL PRACTICAL CONSIDERATIONS

In special situations the anaesthetist is faced with specific problems concerning his patients. Particular plans have to be designed in his head to face the different problems successfuly.

Diabetic patients use specific drugs and diet regiment and may present for surgery in different forms of treatment. Those who are well controlled require no special preparation if undergoing elective minor surgery. However if, the surgical procedure is major, his treatment with long acting insulin should be converted to soluble insulin, the dose of which should be related to blood sugar estimations and repeated at meal times. If the patient is unable to take oral feeding, glucose infusion is substituted.

If his medical condition is uncontrolled or he comes in emergency, the blood sugar level should be estimated. Soluble insulin should be given s.c.and 5% glucose solution infused. One unit insulin is given for each 2 gm glucose. If ketosis is present, insulin-glucose ratio should be 1 : 1. It is safer for the patient to arrive in the theatre on the hyperglycaemic than on the hypoglycaemic side. Special attention should be given to correct any dehydration or electrolyte imbalance. Four hourly urine testing with periodic blood sugar estimations should be continued in the severe case for 48 hours post-operatively

Patients coming for thyroidectomy present a special problem. None should be operated upon until the thyrotoxicosis has been controlled to the fullest extent. Because these patients tend to be excitable, special consideration must be given for pre-operative

sedation. Phenothiazine drugs have a special application and may be particularly useful for immediate premedication. Although local analgesia is satisfactory for thyroidectomy, general anaesthesia is preferred by all patients. Pre-operative oxygenation of the patient is advisable. Light general anaesthesia is satisfactory so long as the thyroid function is under full control. In partially controlled cases, deeper levels of anaesthesia prevent a marked increase in catecholamine production, which may be harmful to the patient. A little foot-down tilt will assist venous drainage and reduce blood loss. Oxygenation and ventilation should prevent hypoxia and hypercarbia which form the dreadful background of serious cardiac complications. The anaesthetist should watch the vocal cords during extubation and make certain that the recurrent laryngeal nerves are not damaged. The patient should be observed in the immediate post-operative period in case a thyrotoxic crisis or respiratory obstruction develops. The signs of crisis are a rapidly rising pulse rate, pyrexia, sweating, hypertension followed by hypotension and heart failure. Treatment needs antithyroid drugs, iodine, steroids and adrenergic blocking agents. Oxygen should be inspired and hypotermia may be of value. Respiratory obstruction may develop in the immediate post-operative period due to damage of the recurrent laryngeal nerves, laryngeal oedema, tracheal collapse or reactionary haemorrhage, each of which will require urgent treatment.

Hypertensive patients whose arterial blood pressure is controlled and maintained within normal limits are better than those who are not treated. There is no reason to stop the administration of antihypertensive drugs before the operation. The effect of

reserpine, guanethidine and methyl dopa can persist for two weeks or longer after withdrawal and the blood pressure would be less controlled. Controlling the arterial tension prevent its abnormal swings during anaesthesia. Still, the controlled hypertensive patient should receive an utmost care. Pre-oxygenation, smooth induction and maintenance together with rapid replacement of blood loss are essential.

Anaesthesia for patients with cardiac disease requires considerable care. These patients should be fully investigated before surgery. The benefit of the patient from surgery should be balanced against the risks of anaesthesia. As a rule, however, most patients stand anaesthesia well unless they are in heart failure. These patients should receive 100% oxygen for few minutes before induction of anaesthesia. Hypotension, hypoxia and hypercarbia are the hazards that should not be allowed. On the whole, the results depend on the myocardial performance and function rather than the clinical diagnosis of the cardiac disease.

Many patients present to surgery while they are on corticosteroid therapy. These patients may have suprarenal hypofunction caused by exogenous corticosteroids. These patients should receive 100 mg hydrocortisone I.M. at the time of premedication. This dose has to be repeated during surgery if unexplained hypotension occurs and 12 hourly in the first post operative day, then the patient returns to his normal regime of corticosteroid therapy. This plan holds for patients with a past history of steroid treatment within up to one year before surgery.

Patients receiving monamine-oxidase inhibitors for mild psychiatric disorders, should not receive vasopressors or pethidine.

MEDICO-LEGAL ASPECTS

The anaesthetist is but a medical practioner whose standards of conduct and practice do not differ from other doctors. However, he is more liable to litigation because the anaesthetic complications, sould they occur, become rapidly evident. The anaeshetist who does his duty within his capabilities without negligence and uses techniques generally considered reasonable and safe has nothing to fear.

However, the anaesthetist should be quite careful in dealing with patients to avoid errors. He should check the identity of the patient, the type of the operation and the side to be operated upon before inducing anaesthesia. He should be sure about the consent of the patient about any specific procedures carried out as amputation of a limb or doing nephrectomy if necessary. The anaesthetist should check his anaesthetic machine personally and be sure about the oxygen supply and electrical connections. The anaesthetist should check the blood used for transfusion. He should be careful not to induce anaesthesia without the presence of a third person at least, as dreaming under light anaesthesia may occur and can lead to claims of sexual assault. He should be careful in conversation at the time of induction and recovery from anaesthesia to avoid any misunderstanding on the patient side, because audition of the patient is the last sense to be lost at induction and the first to be regained at recovery. The anaesthetist is advised to

observe the position of the patient on the operative table, on the trolley and in bed to avoid his injury. He safeguards himself if he writes or fills an anaesthetic record and writes details about a mishap should it occur. The anaesthetist should visit his patient or be in touch with him within the first 24 hours to know how to defend himself in case a complication arises in the early post-operative period and also to give his advice if this is useful for the patient by any means. If the anaesthetist has an insurance to guard him against the hazards of his job, he has to inform the insurance company about the hazard in time. In case any complication takes place, the anaesthetist should not make much discussions with friends, patients relatives or other personnel in the hospital. The careful consideration of these advices puts the anaesthetist on solid legal grounds and proves his clinical care of his patient.

II — CONSIDERATIONS IN PRACTICE OF RESUSCITATION

THE ANAESTHETIST AND RESUSCITATION

The basic principles of resuscitation to tide a patient over a specific period of a danger threatening his life are similar. Physicians and general practitioners deal with different conditions needing resuscitation. The anaesthetist, in particular, is mostly faced during his continuous practice by particular conditions for resuscitation. He has to be experienced in management of shocked patients, apparently dead patients with cardiac arrest, patients with head injury and patients with acute drug intoxication. In all patients needing urgent resuscitation, the anaesthetist should support the cardiovascular and ventilatory functions, deal properly with brain oedema and test and improve the renal function.

It is sufficient for anaesthetists in our country to depend upon their continuous observation and clinical assessment of important parameters as blood pressure, pulse rate and volume, tidal and minute volumes of respiration, calculating urine flow and doing a daily fluid chart. No monitoring system, whatever sophisticated, can replace the meticulous clinical assessment of the patient. However, it may be within the experience range to add two parameters as adjuvants for better management of acute conditions needing resuscitation. These are central venous pressure catheterization and blood gas estimation which can be achieved by direct reading from the blood gas analyzer after its feeding by a small blood sample.

Central venous Catheterization :

By central venous catheterization is meant the location of the tip of a catheter in the superior vena cava, right atrium or inferior vena cave, the superior vena cave being the ideal site.

Central venous catheterization can give valuable informations and can be used for useful applications. It is used for measurement of central venous pressure It can be the ideal rout for parenteral feeding. It facilitates the infusion of drugs near their targets as the administration isoprenaline or dopamine in heart failure. Injection of radio-opaque material for angiography can be achieved through a central venous catheter. A Swan Ganz catheter is inserted through a central venous catheter for measurement of pulmonary capillary wedge pressure. A central venous catheter is used for the measurement of cardiac output by dye dilution or thermal methods.

Central venous pressure measurement is one diagnostic tool in acute circulatory failure and is one guide for fluid replacement in hypovolaemia. Normal values for central venous pressure are 4—12 cm H_2O. Values below this range may indicate hypovolaemia and values above this range may indicate fluid overload or heart failure.

In clinical practice, the anaesthetist can correlate central venous pressure and arterial blood pressure readings to get valualbe information. Low central venous pressure (empty neck veins) with low peripheral arterial blood pressure denote the need

of transfusion of blood or infusion of fluids to fill the circulatory bed. High central venous pressure (full neck veins) with high peripheral arterial blood pressure readings denote overloading of the circulation. Low central venous pressure and high peripheral blood pressure may denote peripheral pooling of the blood. High central venous pressure and low peripheral arterial blood pressure readings denote a block in the myocardial pump, a failing heart. Measurement of urine flow correlated with these findings can make the clinical picture of the patient more clear.

Venepuncture for central venous catheterization can be done in the antecubital fossa, the subclavian vein or the internal juglar vein. Although the subclavian vein is the easiest site, there is a higher risk of early thrombophlebitis due to the small calibers of veins and some risk of pneumo- or haemo-thorax or injury to the brachial plexus or phrenic nerves. Although the infraclavicular approach through the subclavian vein is the best site for long-term catheterization due to the easy fixation, there may be an increased risk of pneumo-thorax. The internal juglar vein gives the most consistent results for correct placement of the catheter and is the most satisfactory rout for catheterization. The right internal juglar vein is easier to cannulate than the left as the area of the thoracic duct is avoided.

Central venous catheterization is not without hazards, but if care and skill are excercised, serious complications become rare and patient's benefit for diagnosis and treatment is great.

MANAGEMENT OF A SHOCKED PATIENT

It is the duty of the anaesthetist to give a hand in the management of different states of patients. But sometimes he is confronted by the double responsibily of resuscitating the shocked patient before giving him anaesthesia. Sometimes, he should deal with a condition of shock originating during the course of surgery. In order to fulfil the responsibility successfully, a sound understanding of the physiology, pathology and biochemistry of shock is indespensible.

Shock is a state of progressive circulatory failure in which the cardiac output is insuffient to maintain tissue requirements of nutrition, oxygenation and waste disposal.

Shock originates from any condition that may lead to a final common path of inadequate tissue perfusion. Exemples are countless including blood, plasma and water loss, obstruction of big vessels, supra-renal insufficiency, nerve trauma and tissue autolysis.

Shock is classified according the causative factors into hypovolaemic, cardiogenic or septic entities. In hypovolaemic shock, the presenting pathology is hypovolaemia due to blood, plasma or water loss. In cardiogenic shock the presenting pathology is an affection of the cardiac pump due to myocardial infraction, pulmonary embolism, arrhythmias, temponade or as a complication of spinal or general anaesthesia. In septic shock, cells of vital organs fail to perform the normal metabolic functions inspite of the availability of oxygen. The anaesthetist is mostly confronted

with shocked patients originating from blood loss before or during surgery. He may deal with shock states of cardiogenic nature during or after surgery.

When shock is due to blood or fluid loss the warm, dry, pink person with a normal pulse and arterial blood pressure becomes cold, moist, pale with a rising pulse rate and falling blood pressure. In such a situation, shock is not diagnosed by one sign or symptom but rather by the syndrome of pallor, restlessness, intense peripheral vasoconstriction, tachycardia with or without hypotension and with or without an increase in the central venous pressure. The intense peripheral vasoconstriction leads to spasm in precapillary sphincters controlling the microcirculation and to opening of arterio-venous shunts. Stagnation of blood occurs in the microcirculation with accumulation of humoural factors and dissiminated coagulation. With progress of the case, the pre-capillary sphincters are paralysed leading to rapid ingress of blood to the microcirculation. The hydrostatic pressure rises in the microcirculation with infiltration from capillary walls and oedema of tissues and liberation of toxins.

When the patient loses blood, he passes through a stage when his compensatory mechanisms are at full to maintain the arterial blood pressure. This is a compensated stage of shock and the microcirculation is affected with a decrease in tissue perfusion. When more blood is lost, the compensatory mechanisms begin to fail and a decompensated stage of shock occurs where the blood pressure falls and tissue perfusion is more impaired. The two stages are early and late components of one and the same syndrome. In

addition, the myocardium itself suffers the same pathology, becomes flabby and potentiates the progress of shock.

Shock affects the respiratory functions of the patient. The physiological dead space increases Maldistribution occurs and more arterio-venous shunts in the lungs open. Respiratory insufficiency takes place specially in patients with a lung disease. Many factors contribute in the adult respiratory distress syndrome.

Shock is a generalised pathology which affects mostly each cell. Other vital organs share the cardiovascular and respiratory systems. Concerning the kidney, changes up to acute tubular necrosis with oligurea or anurea may take place as the kidney cannot withstand prolonged low perfusion. On the other hand, the liver has a wide safety margin and liver function tests may be normal even late in shock. Glycogen stores are depleted then hyperglycaemia is followed by hypoglycaemia with progress of shock. Brain shares the insult and oedema of its tissues is manifested as apathy, drowsiness followed by coma. Endocrinal system participates in the mechanism of shock as catecholamine release is responsible for the spasm of the pre-capillary sphincters. The high catecholamine blood level is responsible for the peripheral vasoconstriction and some of t he metabolic effects. Insulin is partially inhibited. Aldosterone and antidiuretic hormone levels rise during shock to preserve the intravascular volume. The biochemical changes in shock include an increase in lactates and pyruvates, an increase in potassium level due to catabolism, an increase in arterial carbon dioxide tension, a decrease in arterial oxygen tension and a decrease in pH.

The anaesthetist managing a shocked patient should aim at increasing tissue perfusion, and managing the aetiological factors simultaneously. The first and most important rationale of treatment includes the rapid and adequate restoration of an effective circulatory volume. This is achieved by blood transfusion, plasma, plasma expanders and other fluids. When blood is lost, it is a good policy to give the patient lactated ringer in addition to blood to fulfil haemodilution and improve the microcirculation which is a possible site of dissiminated coagulation. So long as we aim at tissue perfusion, it is logic to administer alpha-adrenergic blockers which cause vasodilatation and not vasoconstrictors which increase the peripheral resistance and put an afterlood over the heart which may lead to its failure. The important point is that vasodilators are not administered before compensating blood and fluid loss. Phenoxybenzamine can be given as 1 mg/kg dose repeatedly. This alpha blocking agent specificaly blocks the alphareceptors in visceral and cutaneous vessels. Its action persists for 24 hours. It has antihistaminic and antiserotonin effects, against the endogenously produced amines playing an important rôle in shock. In addition it has a mild analeptic property. Chlorpromazine hydrochloride given in small repeated doses and corticosteroids given in big doses induce alpha-receptor blockade. Corticosteroids act also against endotoxins and improve the cell function. Oxygen administration helps in the management of shock. Morphia if needed should be given intravenously to act at the proper time. Broad-spectrum antibiotics act against endogenous toxins.

The anaesthetist should not depend only on drugs during management of shock. Measurement of central venous pressure

may be helpful. He can use his clinical sense and look at the neck veins instead. When the central venous pressure is low (empty neck veins), it is better to give blood or plasma expanders. When the central venous pressure is low and the haematocrite value is high, it is better to give dextran of low viscosity.

The urine flow can give valuable information during management of shock. The bladder is evacuated by a sterilized catheter. Kidney function is tested by giving 25 gm mannitol I.V., and producing urine at a rate more than 0.5 ml/minute.

Administration of anaesthesia to a shocked patient needs utmost care. Resuscitation should aim at cystolic blood pressure of 100 mm.Hg. Only life saving operations are permitted. Some patients will be saved only if they are receiving anaesthesia and undergoing surgery on the spot like those with ruptured ectopic pregnancy and in those cases no time should be lost as clamping of bleeding vessels is essential. As a rule, general anaesthesia is preferred if local infiltration anaesthesia does not suffice. Patients should be preoxygenated before induction of anaesthesia. Only a sleeping dose of thiopentone 2.5% solution is given. Ketamine is preferred in such cases as it raises the arterial blood pressure. Light anaesthesia is sufficient in shocked patients as they consume small amounts of anaesthetics. Careful observation of respiration, pulse and blood pressure is essential during and after surgery. Correction of fluid status and acid-base imbalance is necessary.

MANAGEMENT OF CARDIAC ARREST

The anaesthetist may be faced with the occurrence of cardiac arrest during a surgical operation or in the post-operative period. He may be, also, urgently asked for cardio-pulmonary resuscitation of a patient in the medical ward He should know the pathogenesis of the condition and the steps of resuscitation in order to achieve success.

If the circulation of a patient fails to such an extent that his brain stops to function, then cardiac arrest is said to occur. This does not apply to conditions caused by irreversible lesion. So, cardiac arrest can be defined as failure of the heart to maintain an adequate cerebral circulation in any situation other than that caused by progressive and irreversible disease.

For proper management of cardiac arrest, the condition should be diagnosed within 30 seconds. Diagnosis depends on absence of pulse in big vessels as the femorals and carotids together with the presence of possible signs of cerebral hypoxia, namely, unconsciousness, apnoea with or without cyanosis and widely dilated pupils. The anaesthetist should not lose time by auscultating the heart or monitoring its function by an electro-cardiogram (E.C.G.). There is no substitute for a rapid clinical diagnosis, if emergency treatment is to be instituted promptly and effectively.

The first duty of the anaesthetist is to produce an effective ventilatory and circulatory function. The airway should be cleared from regurgitation, vomitus or false teeth. Artificial ventilation by 100% oxygen through an endotracheal tube and external cardiac

massage should be done simultaneously. Every 1-2 lung inflations are alternated with 6-8 compressions. Until intubation and oxygen are available, mouth to mouth or mouth to nose ventilation is done. For external cardiac massage to be effective, the patient should be placed on a reasonably hard surface. Compression should be efficient to allow the sternum to be depressed for 3-6 cm then released. External massage aims to regain peripheral pulse and a systolic blood pressure of 60-70 mmHg. This is accompanied by constriction of the pupils and may be the return of respiration and/or consciousness.

An E.C.G. is done, when asystole or ventricular fibrillation is diagnosed and treated accordingly.

If external cardiac massage fails, internal massage is done, which is rarely needed. Internal massage is also thought of if there is a possibility of cardiac temponade or intrathoracic bleeding or if the chest is deformed.

If E.C.G. diagnoses asystole, the anaesthetist continues vigorous external massage. Myocardial tone is increased by the intravenous or intra-cardiac injection of 5 ml of 10% calcium chloride solution and 5 ml of 1/10.000 adrenaline solution. Both drugs may be repeated after 5—10 minutes. Sodium bicarbonate 8.4% solution is given in a dose of 150—200 mEq. I.V. but never intracardiac. These measures usually result in spontaneous heart beat. But if asystole persists, internal cardiac massage is resorted to. If activity returns as ventricular fibrillation, the heart should be defibrillated.

If ventricular fibrillations are poor or slow, calcium chloride or adrenaline is used to make them brisk so as to respond better to defibrillation. Direct current (D.C.) defibrillation is applied as 100-400 joule for external and 30—100 joule for internal defibrillation. Adrenaline can be repeated followed by shock application. If unsuccessful and fibrillation waves are coarse I.V. lignocaine 1 mg/kg is given followed by shock application. If this fails to maintain stable rhythm, propranolol is given in a dose of 1—5 mg, very slowly I.V. and the shock is repeated.

The management of cardiac arrest is never complete if cerebral oedema is not treated to guard against permanent cerebral damage. Frusemide, 20 mg I.V. can be given as an emergency measure. Dehydration is also achieved by giving 25% mannitol Hypothermia, can also be used on occasions. Serious metabolic acidosis requires equally urgent treatment. Renal function should be tested by a small mannitol challenge and dialysis is resorted to should renal failure has taken place.

MANAGEMENT OF HEAD INJURY

Craniocerebral injuries may be manifested in the form of a scalp laceration, a skull fracture, a cerebral concussion, contusion or laceration with or without etradural, subdural subarachnoid or intracerebral haemorrhage. In addition patients with head injuries may have injuries as well in other parts of their bodies.

At the time of injury, although a complete neurological examination is useful, a definite diagnosis is always difficult. The most

important determinations to be established at that time are :

— The presence of shock.

— The adequacy of the airway.

— The presence of multiple injuries specially spinal cord injury.

— Evidence of increasing intracranial pressure

Priority should be given to the first two items if proper management is aimed at.

SHOCK :

A severe head injury alone rarely causes shock except if profuse bleeding from an extensive scalp laceration has taken place. Based on this, the presence of shock usually points to associated multiple injuries. The examiner should look for ruptured abdominal organs, intra-thoracic bleeding or fractures specially in the pelvis or femur. The treatment of shock is given the priority even if the patient is unconscious except if it is clear that the patient suffers from an extradural haematoma. Shock should be treated in situ before transferring the patient for any reason, and no movement of such a shocked patient is allowed. The early diagnosis of an associated spinal cord injury is important to avoid further cord damage. In the comatose patient, this is proved by absence of deep reflexes in the lower limbs and lack of response to painful stimuli in the lower limbs while response is present in the upper limbs.

THE AIRWAY :

The respiratory passages may be obstructed by blood, secretions or vomitus. The lowered head should be positioned on the lateral side with finger scoobing of the cheeks and suction to clear the upper respiratory passages. In the comatose patient with respiratory difficulty endotracheal intubation may be needed and tracheotomy may be life saving.

Intracranial Pressure :

A patient with a closed head injury should be put under observation to determine if treatment is surgical or medical. This depends on signs of increasing intra cranial pressure due to the presence of intracranial haematoma or the development of brain oedema. The most important parameters to be monitored are the level of consciousness, the blood pressure, pulse, respiration and pupil size. These should be recorded every 15 minutes for 1—2 hours then every 30 minutes and every 60 minutes thereafter depending on the particular case. An acute increase in intracranial pressure is safely diagnosed by a decreasing level of consciousness, a rising blood pressure, a slowing pulse rate and irregular respiration. If pressure continues to rise, the patient may become comatose. Unilateral dilatation of the pupil followed by fixation to light indicates a third cranial nerve compression. Bilateral constriction and fixation of pupils indicate pontine involvement.

MOTOR FUNCTION :

Rising intracranial pressure may cause changes in motor function in the form of increasing hemiparesis, increased deep tendon reflexes and positive Babinski's sign.

ELECTROLYTES :

Serum electrolytes should be checked every day. Electrolyte imbalance after cerebral injury may produce drowsiness, coma and convulsions, which may complicate the picture of increasing intracranial pressure. Hyponatraemia due to faulty scheme of parenteral therapy causes an increase in intracranial pressure which may be fatal specially in early management.

Treatment of increased intracranial pressure :

After checking the renal function, mannitol in a dose of 1—2 gm/kg is given over 30—60 minutes. In the comatose patient a sterile urinary catheter is used to calculate the renal response to mannitol. Treatment in every case depends upon the response of the patient during the observation period.

Supportive treatment :

The anaesthetist can share to tide the patient with head injury over the period of depressed vital functions. A patient may get spontaneous cure from the head injury within few days but succumb from failure of one vital function. Adequate care should be given to the chest, cardiovascular system and kidneys. Infection should be guarded against and excellent nursing is invaluable to guard against and cure bed sores, chest and urinary infection. Nutrition in the conscious and comatose patient is given enough care. Volume, concentrational and caloric deficits should be compensated in all patients. Bed side chart and monitoring is necessary in every patient with head injury even if the injury is considered

trivial. The prognosis of all patients depends on the meticulous observation and wise management.

MANAGEMENT OF THE ACUTELY POISONED

The management of acutely poisoned patients requires active interferences which are a part of the clinical practice of the anaesthetist. Whether patients are poisoned due to accidental exposure to insecticides or due to trying to commit suicide, may not alter the essential requirements for management significantly.

Most of the patients can be successfuly treated with good supportive therapy after accurate assessment of their condition.

Assessment .

The vital functions of patients should be rapidly assessed before losing time in diagnosing the cause of poisoning. Attension should be paid to assess the central nervous system, the cardiovascular system, the respiratory system and the body temperature.

Assessment of the central nervous system :

Most of the patients poisoned by large doses of drugs go into coma, the depth of which provides a good guide for evaluating and following up their conditions. A simple scheme for grading coma can be easily applied clinically. In grade 1, patients are drowsy but they respond to verbal commands. In grade 2, patients are unconscious but they respond to minimal stimuli. In grade 3, they

no longer respond to minimal stimuli, but to maximal painful stimuli. In grade 4, the unconscious patients are totally unresponsive and electro-encephalography is needed for the follow up.

Assessment of the cardiovascular system :

Monitoring of arterial blood pressure, pulse rate and rhythm and central venous pressure is essential for the cardiovascular assessment of the poisoned patient. But the clinical observation of the patient gives the fundamental parameter for assessment. It gives a good idea about the degree of tissue perfusion of the patient. The well perfused patient has warm extremities with dilated non-empty veins, a pink colour with no central or peripheral cyanosis and passes urine at a rate more than 0.5 ml/minute. In addition his systolic blood pressure is above 80 mmHg although, a hypertensive crises may be met with when a monoamine oxidase inhibitor is the cause of poisoning.

Assessment of respiratory system :

Assessement should include the patency of the airway, the presence of the protective cough reflex, cyanosis, the minute volume of respiration and arterial blood gases. The tidal volume can be measured using Wright's respirometer and a face mask. A reduction in the minute volume of respiration of 50% denotes inadequate tidal exchange, and necessitates the measurement of arterial blood gases. An arterial oxygen tension below 60 mmHg and carbon dioxide tension above 55 mmHg indicate the urgent need for artificial ventilation.

Assessment of body temperature.

Most of acutely poisoned patients are hypothermic due to impaired tissue perfusion. A core temperature below 36°C denotes that the patient is in a serious condition. In addition the comparison of simultaneously recorded core and shell temperatures gives an idea about the haemodynamics of the patient. The core temperature is measured from the nasopharynx, oesophagus or rectum. The shell temperature is measured from the skin. When core and shell temperatures are low, they denote hypothermia. In the same way if both are high, they denote a febrile reaction of the patient. It is interesting to understand that a constant or rising core temperature coupled with a falling skin temperature, denote falling tissue perfusion and in particular peripheral vaso-constriction.

After assessment of the vital functions of the patient is completed, it may be of value to determine the cause of intoxication. Samples of blood, urine and gastric contents should be sent for the laboratory for analysis. Knowing the cause of acute poisoning may help in the management if a specific antidote is available.

Management :

Management of the acutely poisoned patient is essentially a supportive therapy of his vital functions, removal of the poison from his body and the antagonism of its effects by antidotes if felt valuable.

Supportive therapy for the cardio-vascular system :

The acutely poisoned patient has a reduced cardiac output due to a relative degree of hypovolaemia as the poison induces some-

expansion of the microcirculation. Elevation of the legs is a useful short-term measure to improve venous return. The administration of vasopressors increases the risk of hepatic and renal ischaemia and the administration of intravenous plasma expanders is more useful. Lactated Ringer, 5% dextrose and 0.9% sodium chloride are given according to the urine output. Diuretics may be used to test the renal function or to augment the renal excretion of the poison.

Supportive therapy for the respiratory system :

Patients should be positioned on their left side where secretions, vomitus and saliva are aspirated followed by putting an oropharyngeal airway in order to maintain a clear airway. Any doubt concerning the patency of the airway should urge the anaesthetist to put a cuffed endotracheal tube under direct vision laryngoscopy. Oxygen should be administered to correct any hypoventilation or cyanosis. The follow up of the condition needs repeated measurement of arterial oxygen and carbon dioxide tensions in the blood. Inspired oxygen should be always warmed and humidified Care should be taken as the use of ventilators may be associated with marked falls in the cardiac output. Tracheostomy is sometimes needed and bronchial toilet is repeatedly done through the tracheostomy or the endotracheal tube.

Removal of the poison from the body :

When more than four hours have passed since the ingestion of the poison, there is no point in attempting to empty the stomach.

Evacuation of the stomach should be done by using a wide bore gastric tube. Care should be taken whenever the protective cough reflex is lost. It is a good policy to evacuate the stomach while patient's head is low and he is on his left side.

Drugs, nursing and other measures :

In some patients, the use of specific antidote may be life-saving. The use of analeptics have no place in management of the comatose although doxapraram is said to give good results. Some patients benefit from haemodialysis in management of renal failure. Diazepam may control convulsions Most patients can be rewarmed by blankets and heated matresses to control hypothermia. Nursing should include the follow up chart of fluid balance and the progress of vital signs of the patients

III — SOME THEORETICAL CONSIDERATIONS

IN

ANAESTHESIA AND RESUSCITATION

BODILY RESPONSES TO ANAESTHETIC TRAUMA

The trem «milieu interieur» was coined by Bernard to mean the composition of the body fluids constituting the body intermal environment.

The term «homeostasis» was coined by Cannon meaning constancy of the internal environment after its distortion by external stresses. This occurs through autoregulation towards normal after disorders that acutely threaten life. The Bernard — Cannon concept has been achieved through a variety of biochemical, endocrinal and metabolic alterations. These changes follow any injury in the form of simple blood loss, fractures, burns, surgery, anaesthesia, acute illness or even psychological trauma. The different changes after any injury or trauma are :

1 — Body cell mass loss.

2 — Conservation of extracellular fluid (E.C.F.)

3 — Changes in energy source.

4 — Changes in cardiac output.

5 — Changes in renal function.

6 — Changes in pulmonary function.

7 — Changes in the gastro-intestinal tract.

8 — Changes in cerebral function.

9 — Endocrinal changes.

10 — Changes in the fibrinolytic activity of the blood.

1. Body cell mass loss :

After injury lysis of cellular protoplasm takes place with release of cellular products into E.C.F. some compounds are converted into glucose which is burnt in the normal way. Most of the nitrogen is excreted as urea in urine. Skeletal muscle is mostly affected as shown by increased creatine and creatinine levels in blood and urine respectively and also by the fairely rapid decrease in the bulk of palpable muscles. Transient immobility and starvation after injury add to the muscle insult. Intracellular electrolytes particularly potassium, phosphate and sulphate are lost, through cellular lysis, into E.C.F. and hence to urine via the kidney. In spite of this, skeletal muscle recovers completely without residual weakness. Various visceral organs as brain, heart, lungs, kidney and liver suffer this post-traumatic catabolism with less severity. By contrast, prolonged post-traumatic starvation has severe adverse effects upon all bodily functions and is corrected by increased oral or parenteral intake.

2. Conservation of E.C.F. :

After injury the body adopts several active mechanisms to conserve E.C.F. so as to maintain plasma and blood volume. The body concentrates on sodium conservation as the total osmolarity of E.C.F. and therefore its total volume is largely determined by the sodium content. Sodium is the water holding ion of E.C.F. Its conservation occurs through its reabsorption mainly in the distal convoluted tubules and partly through the distal small bowel. This is manifested by a decreased absolute sodium excretion rate, a

decreased sodium concentration in urine, inability to excrete sodium bicarbonate, a tendency towards aciduria and a decreased sodium content of saliva and sweat. This is accompanied by reduction in water loss in urine, saliva, sweat and gastro-intestinal juice.

The plasma concentration of sodium and potassium move in opposite directions and at similar rates. When sodium is retained, potassium and hydrogen ions are lost, or move into cells, with tendency of extracellular alkalosis. When sodium is lost, potassium and hydrogen ions are retained with tendency of extracellular acidosis and hyperkalaemia.

After injury patients retain sodium and water more easily when they receive sodium than in the mormal state. Dilutional hypoproteinaemia occurs with tendency towards oedema in tissues including the lung, brain and peripheral areas. Also, large water load in the post-injury period leads to water retension through an antidiuretic mechanism. So, moderation in the administration of sodium or water after trauma is the best policy if adverse effects are to be guarded against

3. Changes in energy source :

After injury the body is faced with a state of acute starvation. So the normal energy source of mixed exogenous diet is shifted to endogenous fat oxidation. After depletion of glycogen stores in the first few hours, fat provides most of the patients energy requirements. Fat is mobilized from depot fat by hydrolysis to free fatty acids and glycerol. Muscles can burn fatty acids directly while other tissues utilize them after more degradation in the liver.

When blood glucose concentrations are low and insulin secretion is thus inhibited, the body releases amino acids from muscles which are converted to glucose. So, mobilization of muscle protein provides some energy. After trauma there is a small and persistent rise in blood sugar unaccompanied by an appropriate rise in plasma immunoreactive insulin together with a rise is plasma free fatty acids.

4. Changes in cardiac output :

Provided the patient is resuscitaed after trauma or operation or following a low flow state, an elevated cardiac output occurs in the post-traumatic period. This is due to the effect of catecholamines on the myocardium and aims to meet the increased tissue demands of blood when the oxygen — dissociation curve is shifted to the left. It also meets increased work of breathing, increased temperature and sepsis.

5. Changes in renal function :

After trauma with volume or flow challenge, differential renal vasoconstriction occurs with hypoperfusion of the kidneys. This is associated with shunting of blood away from renal cortex, decreased filtration and maximal tubular reabsorption due to increased secretion of aldosterone and antidiuretic hormone. These factors may end in post traumatic renal insufficiency. The risk of renal damage increases when some degree of renal disease is present before trauma.

Adequate hydration and solute loading are protective to the

kidney after trauma to maintain well-filled renal tubular laminae and high glomerular filtration rates. This is achieved by fluids as mannitol, glucose or salts.

6. Changes in pulmonary function :

After trauma, the integrity of the lungs is threatened by various factors. Direct trauma to the thoracic wall with flial chest and collapsed or under-perfused lung tissue and aspiration of gastric contents are among these factors. Fluid overload with pulmonary oedema, multiple small emboli from the trauma site, or banked blood, micro-organisms from peripheral infection and vasoactive substances as catecholamines, serotonin and kinins are complicating factors. Post-traumatic pulmonary insufficiency occurs when the resultant of these factors overbalances the tolerance of the particular lung. Lung insufficiency always follows when there is a history of an obstructive lung disease, namely, chronic bronchitis, bronchial asthma or emphysema. This is similar to the adult respiratory distress syndrome. Hyperventilation with hypocarbia, respiratory alkalosis and prolonged anoxaemia not corrected by oxygen therapy are charecteristic findings of post-traumatic pulmonary insufficiency.

7. Changes in the gastro-intestinal tract ;

After severe trauma the power of absorption and propulsion of the gastro-intestinal tract is reduced. This explains abdominal distension and vomiting which accompany early feeding after trauma. If trauma is long standing as in burns, pulmonary insuffi-

ciency or myo-cardial infarction, acid-peptic auto-digestion of the stomach and duodenum occurs in the form of stress-ulcer. Severe haemorrhage from the upper gastro-intestinal tract may be the terminal finding.

8. Changes in cerebral function .

After severe trauma, metabolic encephalopathy occurs due to inadequate perfusion or oxygenation of the brain. Hypocarbia or alkalosis or both produce cerbral vasoconstriction. Disorientation, hallucination, restlessness and coma follow. As glucose is not at short after trauma, lack of oxygen and improper perfusion are the limiting factors.

a) Endocrinal changes :

After severe injury or prolonged low flow states, the pituitary and adrenals may become either hyperaemic or infarcted. Sheehans syndrome involves pituitary infarction during haemorrhage associated with parturition. Adrenal apoplexy involves haemorrhage into the adrenals sometime seen after severe burns. Prolonged administration of corticosteroids produces atrophy of the adrenal cortex which is symptomatic if the patient is injured or operated upon. Haemorrhage in the adrenal cortex with adrenal failure may occur in patients under antico-agulant therapy or after localization of blood born bacteria in the adrenal cortex. Although these affections are very rare, they do prove that endocrine glands may be affected following severe trauma

A rise in blood cortisol level is charecteristic aftar trauma. If

the injury is self — limited, the level quickly falls to normal. If the injury is long-standing or if infection maintains the traumatic status, the level remains high for weeks or months.

Isotonic volume reduction as in simple blood loss is a potent stimulus to aldosterone secretion. This stimulus is activated through the secretion of renin by the juxtaglomerular apparatus of the kidney in response to decreased renal blood flow. This in turn stimulates the production of angiotensin, which directly stimulates the adrenal cortex to produce aldosterone. A.C.T.H. is also a potent stimulator of aldosterone production. A decrease in plasma sodium concentration, a decrease in extracellular volume, or an increase in plasma potassium concentration may stimulate aldosterone production.

Aldosterone leads to sodium conservation to support the plasma volume. A decreased renal excretion of sodium bicarbonate in an acid urine containing increased amounts of potassium is characteristic of the post — traumatic state.

An isotonic reduction of blood volume stimulates the production of vasopressin (antidiuretic hormone) from the supra — optic tract and its release from the posterior pituitary. If plasma is hypertonic, vasopressin production is further stimulated. If the patient is treated by sodium-free water, hyponatraemia will develop due to vasopressin water retension. When water is taken by mouth and the plasma hypertonicity is restored to normal, antidiuresis ceases promptly.

Increased catecholamine secretion is considered the main en-

docrine response to trauma. Catecholamines have various effects including stimulation of the pituitary producing A.C.T.H. and therefore glucocorticoids and aldosterone. Catecholamine production causes a slightly elevated blood sugar level, a lowered immuno — reactive insulin level and a gradually rising concentration of glucagon in the blood.

Athough growth hormone is known to increase after trauma, thyroid hormones do not take a particular pattern.

After trauma there is decreased libido in the male and cessation of menses in the female suggesting that gonadal steroid activity is reduced.

10. Changes in the fibrinolytic activity of the blood :

Changes in blood fibrinolytic activity accompany surgical interference. Post-operative changes are biphasic. An initial increase in activity is followed by a period of reduced activity termed fibrinolytic shutdown which may persist for the first post-operative week or longer.

Under normal conditions, there is a constantly dynamic equilibrium between the coagulation and fibrinolytic systems. When it is upset by various stresses including anaesthesia and surgery, disturbances occur. A predominant coagulation system causes thrombosis, while a predominant fibrinolytic system causes oozing of blood. Predominant hyper-coagulability of the blood is attributed to sympatho-adrenal stimulation. Predominant enhancement of fibrinolysis is, also, thought to be a stress-induced physiologi-

cal reaction affected by pituitary-adrenal hormones. On the whole, anaesthetic and surgical trauma are responsible, through the changes they induce in the fibrinolytic activity, for the possible occurrence of post-operative deep venous thrombosis.

It is emphasized that all the above mentioned changes may follow anaesthesia. Surgery produces more severe changes than anaesthesia, but, the anaesthetist administer anaesthesia to a surgical patient. Improper management of the surgical patient and/or badly conducted anaesthesia may lead to more severe body responses that affect the life of the patient.

BLOOD TRANSFUSION

Blood is frequently transfused when minor blood losses have taken place. However, each transfusion exposes the patient to potential risks. Based on this, blood should be transfused for certain indications. The major indications are to restore blood volume and or to improve the oxygen carrying capacity of the blood. A haemactocrit of 30% or a haemoglobin of 10 gm % can insure an adequate tissue oxygen supply. In such situations, plasma volume expanders are preferably infused instead of blood. Some trials are undertaken to reduce the needs for homologous blood. Autologous blood transfusion is an exemple in which patients receive their own previously donated blood in elective surgery.

Frozen blood can solve the problem of difficulty in continuous blood supply from blood banks. Blood may be frozen and stored for long periods at very low temperatures of — 80°C to -190°C. The technique includes adding glycerol to freshly collected blood af er removing the plasma to protect the red cells during freezing and thawing processes.

Advantages of frozen blood.

1 — Red cell supply for transfusion is increased.

2 — Rare blood types are preserved in sufficient quantities for time of need.

3 — Blood is collected and reserved for patients who insist on autotransfusion.

4 — As frozen blood is free from white cells and platelets, it is ideal for transfusing patients during cardio — pulmonary bypass and for transplantation recepients.

5 — Frozen blood seems to be free from viable hepatitis viruses.

Disadvantages of frozen blood :

1 — Special freezing, thawing and storage facilities are needed.

2 — Limited stability of red cells after thawing.

3 — Absence of coagulation factors, platelets and white cells.

Blood components :

By utilizing double, triple or quadruple blood collecting sets, a single unit of blood can be analysed for its components which are :

1 — Packed red blood cells.

2 — Platelet rich plasma :

The first collecting set containing whole blood is centrifuged at a speed which produces packed red cells and supernatent platelet rich plasma.

3 — Platelet concentrate : The platelet rich plasma is centrifuged at high speed and the concentrate so seperated is a platelet concentrate.

4 — Cryoprecipitate : the seperated supernatent platelet poor plasma is frozen at — 80°C and thawed at 4°C. Another high speed centrifugation seperates the cryo-precipitate from platelet poor plasma.

The specific blood component recessary for certain patients can thus be safely used. For exemple, packed red cells are preferred for patients with renal or hepatic insufficiency to reduce the amounts of potassium, ammonia and excessive acids that are infused. Packed red cell transfusion improves oxygenation without overloading the circulation. Platelet transfusion controles bleeding in patients with platelet deficiency. Because platelets cannot be stored, appropriate platelet concentrates are prepared as needed.

Complications of blood transfusion :

1. Haemolytic reactions : As little as 25 ml or as much as 500 ml of transfused blood may cause a haemolytic reaction. Most reactions occur during the early period of transfusion, but all recepients should be carefully observed during the whole transfusion time. The clinical criteria of a transfusion reaction include fever, chills, flushing, headache, tight constricting pain in the chest, breathlessness, nausea and pain in loins. Vomiting, diarrhea, haemoglobinuria and oozing from wounds may follow. Comatosed or anaesthetized patients show unexplained tachycardia, hypotension or bleeding.

Once a haemolylic reaction is suspected, the transfused blood is discontinued. Measures are taken to correct hypotension, to control haemorrhage and to prevent anuria. Supporting circula-

tion and respiration is achieved by vasopressors and oxygen. When hypotension is corrected, haemorrhage most probably stops as the state is self — limiting. The insult to the kidneys requires prompt therapy to prevent or minimize tubular necrosis and anuria. This is achieved by correcting hypotension, using bicarbonates to maintain an alkaline urine and by osmotic diuretics as mannitol. If these measures are taken early, tubular necrosis and anuria are mostly prevented. If anuria occurs, acute renal failure is treated with limitation of fluid intake to 400 — 500 ml per day plus the daily output and careful monitoring of plasma potassium, blood urea and creatine. Peritoneal or haemo-dialysis should be performed when needed.

Post-transfusion jaundice may take place after repeated transfusion of stored blood. The old red cells are destroyed with an increase in free haemoglobin and later bilirubinaemia. Usually, no treatment is needed for this condition.

2. Pyrexial reactions : These are the most common side — effects of blood transfusion. Most reactions are assumed to be due to some components of the donor's blood as white cells or platelets. Pyrogenic reactions are markedly reduced by using sterile disposable plastic transfusion sets. Clinically, reactions vary from transient rise of temperature and mild chills to high fever and severe chills, accompanied by headache, nausea, vomiting and muscle pain. Transfusion should be stopped as it cannot be distinguished from an early haemolytic reaction. Most often, no incompatibility is detected and only antipyretics are given.

3. Allergic reactions : They occur in 2 — 3% of transfusions due to allergens in donor's blood. Itching and diffuse rash appear and rarely laryngeal oedema and collapse occur. Treatment consists of stopping the transfusion and giving antihistaminics. The transfusion is continued if treatment is effective and the reaction does not reappear. For severe reactions, steroid preparations are required.

4. Contaminated blood : It may produce chills, fever, general pain, hypotension and shock. Most of the reactions are due to endotoxins of gram-negative bacteria. Rapid administration of antibiotics and steroids together with fluid replacement are life — saving.

5. Microcirculation blockade : Microemboli from the banked blood may accumulate in the lung and contribute in acute respiratory distress of the «shock lung». A 40 — micron mesh filter should be used for each unit of blood in patients with multiple trauma or chronic obstructive pulmonary disease from the prophylactic point of view.

6. Transmission of disease . Malaria, syphilis and hepatitis are readily transmitted via blood transfusion.

7. Hyperkalaemia : Potassium is lost progressively from red cells to plasma. Plasma potassium concentration may reach 40 mEq/L in three weeks old blood. The transfusion of several units of aged blood may produce cardiac arrhythmias and cardiac arrest from hyperkalaemia.

8. Citrate intoxication : Excess citrate in transfused blood will bind some of the recepients calcium. Reduced calcium augments cradiac effects of hyperkalaemia. When the adult patient receives more than two units of blood, calcium gluconate is slowly given in a dose of 10 ml of 10% solution and can be repeated.

9. Cardiac arrest : Massive blood transfusion of old cold blood may cause cardiac arrest due to the effects of hyperkalaemia, excess citrates and hypothermia.

10. Hepatic coma : The progressive increase in ammonia makes old blood unsuitable for patients with hepatic cirrhosis and infusion of several units may precipitate hepatic coma.

11. Other complications : These include complications of any intravenous fluid infusion as air embolism, phlebitis and volume overload.

PRINCIPLES OF FLUID AND ELECTROLYTE BALANCE

Total Body Water (T.B.W.)

Water constitutes 50—70% of total body weight in adults. The highest proportion is found in newborn infants with a maximum of 75—80%. The proportion decreases steadily and significantly with age to 45—55%. Obese persons have less water than lean ones. Females hold less water than males due to more subcutaneous fat. Body water is divided into three functional compartments :

1 — **Intracellular fluid (I.C.F.) :**

Constitutes 40% of T.B.W.

2 — **Extracellular fluid (E.C.F.) :**

Constitutes 20% of T.B.W. and is subdivided into :

a) Intravascular fluid or plasma : 5% of T.B.W.

b) Extravascular or interstitial fluid : 15% of T.B.W.

Chemical composition :

The different compartments, contain cations and anions in chemical equilibrium. The total cations equal the total anions as mEq/L in each compartment. The main cations in the intracellular fluid are potassium (K) and magnesium (Mg) while the main anions are phosphates (PO4) and protein. Sodium (Na) is the

principal cation while chloride (CL) and bicarbonate (Hco3) are the principal anions in the extracellular fluid.

Normal values :

I.C.F. :

Cations . K, 164, Mg, 28, Na, 11, Calcium, 2 mEq/kg

Anions : Po4 : 105, So4 : 20, Protein, 65, Hco3 10 mEq/kg

E.C.F. :

Cations : Na, 142 , K, 4 , Ca, 5 , Mg, 3 mEq/L

Anions : C1, 103 , Hco3, 27 , protein, 16 , So4 : 1, Po4, 2 , Organic acids 5 mEq/L.

E.C.F. can be determined by analysis of plasma and its electrolytes are calculated as mEq/L. I.C.F. is more difficult to obtain because it is always mixed with E.C.F. It can be calculated as T.B.W. — E.C.F. and can be be only termed as mEq/kg.

Units of measurement :

mgm or gm% = the weight of the electrolytes / unit volume. It does not allow a physiologic comparison of the solutes in solution.

Moles or millimoles = The number of particles / unit volume.

Eq. or mEq/L = The number of electric charges / unit-volume.

Osmole or milliosmole = The number of osmotically active particles or ions / unit volume.

Mole = molecular weight in grams

m.mole = molecular weight in milligrams.

$$\text{Equivalent (Eq.)} = \frac{\text{atomic wt.in gm}}{\text{valence}}$$

$$\text{mEq.} = \frac{\text{atomic wt.in mgm}}{\text{valence}}$$

$$\text{mgm \%} \times 10 = \text{mgm/L}$$

$$\frac{\text{mgm/L}}{\text{Eq. wt}} = \text{m.Eq/L}$$

Classification of fluid balance disordess

I — Volume disorders :

If an isotonic salt solution is added to or lost from the body fluids, only the volume of E.C.F. is changed.

So, volume disorders are :

A) E.C.F. deficit, or

B) E.C.F. excess

II — Concentrational disorders :

If water alone is added to or lost from E.C.F., the concentration of osmotically active particles will change. If E.C.F. is depleted of sodium, water will pass into the I.C.F. until osmolarity is again equal in both compartments. Sodium accounts for 90% of the osmolarity of E.C.F.

So, concentrational **disorders are :**

A) Hypo-natremia, or

B) Hyper-natremia.

Sometimes mixed volume and concentrational disorders occur and the signs and symptoms are additive or nullifying i.e. administration of much sodium with restricted water intake leads to hypernatraemia or water deficit.

III — Compositional disorders :

The concentration of most other ions in E.C.F. can be changed without significant change in osmolarity producing only compositional changes which are.

A) Acidosis and alkalosis

B) Hpo and hyperkalemia

C) Hypo and hypercalcemia

D) Hypo and hypermagnesemia

1 — Volume disorders :

A) E.C.F. deficit :

Water and electrolytes are lost in the same proportion as those in which they exist in normal E.C.F. This occurs in :

1) Loss of fluids from the gastrointestinal tract as vomiting, diarrhea, nasogastric suction, draining fistulae and intestinal obstruction.

2) Peritonitis and infections.

3) Burns.

Signs and symptonas :

1) Signs of barbiturate intoxication.
2) Tachycardia and hypotension.
3) Soft wrinkled tongue and sunken eyes.
4) Decrease of body temperature

B) E.C.F. Excess :

due to

1) Iatrogenesis, or
2) Secondary to renal insufficency.

Signs and symptoms :

In the young healthy adult, there are signs of circulatory overload mostly manifested in the lungs. In the elderly patient, congestive heart failure with pulmonary oedema may develop rather quickly with a moderate volume excess.

II — Concentrational Disorders

Sodium (Na)

Sodium intake lies between 50 and 300 mEq/day. Sodium may be lost in sweat according to climate, in stools or urine. Although mineralocorticoids may increase the absorption of sodium from the small intestine and colon, the main burden falls on the kidney as sodium reabsorption occurs mainly in the tubules by a process

consuming energy and oxygen. Aldosterone is responsible for preservation of sodium. It is secreted by a reflex nervous mechanism originating from blood volume receptors or by a humoral rennin — angiotensin system originating from renal ischaemia. Aldosterone also increases the excretion of potassium and hydrogen ions.

(A) Sodium Deficit (Hypo-natremia, Water intoxication)

Causes :

1 — Decreased intake : corrected by tubular reabsorption but aggravates other causes.

2 — Loss in alimentary secretions as vomitus or diarrhoea

3 — Loss in sweat in hot climate.

4 — Loss in urine specially with salt losing nephritis, osmotic diuresis by glucose in diabetic coma or by urea at recovery from acute renal failure.

Clinical picture :

The average sodium level in blood in 142 mEq/L. Acute symptomatic hyponatremia occurs when sodium is less than 130 mEq/L. The picture is characterized by central nervous signs of increased intracranial pressure and tissue signs of excessive intracellular water. Cardio-vascular signs are secondary to increased I.C.P. as bradycardia and hypertension. There is relatively rapid development of oliguric renal failure which may not be reversible if therapy is delayed.

Diagnosis :

Clinically and by laboratory tests.

Treatment :

1 — Minor deficit : Add sodium to diet.

2 — Moderate deficit : I.V. saline 2—3 litres without overloading the circulation. If acidosis is present, part is given as bicarbonate or as lactate.

3 — Severe deficit : To avoid circulatory overloading, hypertonic sodium infusions are given, one fifth of which should be lactated.

(B) Sodium Excess (Hypernatremia, water deficit)

Sodium excess means failure of the kidney to respond normally to sodium load by excerting increased amounts of sodium, due to :

1 — Intrinsic renal disease, or

2 — Inadequate renal perfusion, or

3 — Excessive tubular sodium reabsorption.

Sodium retension occurs in .

1 — Primary aldosteronism.

2 — Cushing syndrome.

3 — Nephrogenic diabetes insipidus.

4 — I.V. hypertonic saline.

Clinical picture :

1 — Restlessness or coma.

2 — Tachycardia and hypotension.

3 — Dry sticky mucous membranes.

4 — Hyperpyrexia.

5 — Oedema.

Diagnosis :

Clinically the condition is diagnosed early. Laboratory testing confirms the diagnosis and is used for follow up of the case.

Treatment :

1 — Restriction of intake.

2 — Cation exchange resins.

3 — Encouraging urinary output as by diuretics.

III — COMPOSITIONAL DISORDERS

POTASSIUM (K)

The daily intake of potassium is 50—150 mEq. Its excretion entails 5—10 mEq/day in the stools and 70 uEq/minute in urine. In renal failure and high potassium intake, there may be tubular secretion of potassium.

Plasma potassium regulation is important as its level affects many physiological functions of the different body systems including neuro-muscular functions.

Potassium deficit (hypo Kalaemia) :

Potassium is lost through the alimentary tract or the kidney.

Alimentary loss : is usually acute and takes place in the following conditions :

1 — Pyloric obstruction or stenosis, where vomiting is associated with semi-starvation.

2 — Aspiration of gastric and intestinal contents when potassium intake is not augmented.

3 — Loss in stools as in ulcerative colitis or after total gastrectomy.

Renal loss of potassium is usually chronic and occurs in the following conditions :

1 — Appropriate renal behaviour towards potassium released from the cells as in diabetic coma or causes of excessive protein catabolism.

2 — Hormonal factors as primary aldosteronism or secondary aldosteronism due to heart failure, hepatic oedema or nephrotic syndrome.

3 — The use of diuretics.

4 — Primary renal disease : generally, there is a tendency to potassium retension but in pyelonephritis, potassium is lost.

Clinically, patients with minor loss show no symptoms. Symptoms appear when potassium level reaches below 3.5 mEq./L. It should be noted that severe sodium loss may be accompanied by

potassium loss. This is to be thought about during infusion therapy.

The cardiac muscle is affected by potassium loss. The ECG shows depressed ST segment, proldonged QT interval, and sometimes inverted T. Digitalis leads to a fall in potassium concentration. Digitalis and hypokalaemia are dangerous.

The skeletal muscles undergo from weakness to widespread palsy and areflexia with respiratory involvement. A common clinical picture of a patient with hypokalaemia includes hypotension, shallow rapid breathing, abdominal distension and mental confusion.

The treatment of hypokalaemia is administration of potassium orally and/or I.V. Potassium concentration should not exceed 40 mEq/L. given as one litre in three hours. It is advisable to monitor the heart by ECG during potassium administration.

Potassium Excess (hyper kalaemia)

Potassium excess is usually associated with catabolic states. Renal insufficiency due to different reasons is usually accompanied by hyperkalaemia.

Infusion of stored blood, trauma, infection and acidosis are other causes.

Clinically, patients exhibit arrhythmias ECG shows peaked T wave in early hyperkalaemia, then prolonged P—R interval, then widened Q—T segment followed by ventricular fibrillation or

asystole. Death occurs before plasma potassium level reaches 10 mEq/L.

Treatment entails stopping ingestion of proteins and increasing carbohydrate diet, promotion of potassium excretion by diuretics, transference of potassium from ECF to the cells by giving glucose and insulin and counteracting cardiac toxicity by measures including digitalis.

ACIDOSIS

Acidosis means excess actual or potential hydrogen in the blood while acidaemia means excessive concentration of hydrion (hydrogen ion) in the plasma. Compensated acidosis means acidosis without acidaemia while uncompensated acidosis means acidosis with acidaemia.

Acidosis may be classified as :

— Metabolic acidosis which entails excessive production of hydrion.

— Renal acidosis which entails defective renal elimination of the hydrion.

— Respiratory acidosis denoting defective respiratory elimination of potential hydrion.

Metabolic acidosis :

It is a condition of base deficit or the excess of any acid other than carbonic acid.

Metabolic acidosis may be due to :

— Accentuation of normal production of hydrion : as in fever, starvation, dehydration or diabetes.

— Administration of acids : as ammonium chloride.

— Loss of alkaline fluid : as the pancreatic juice.

— Lactic acidosis which is encountered in severe circulatory insufficiency, extra corporeal circulation or diabetes.

The compensatory responses to metabolic acidosis include enhansed respiration to lower the carbon dioxide tension, increased excretion of hydrion by the kidneys and the action of buffers.

Renal acidosis.

It occurs due to impaired ability to excrete acid phosphate (H_2PO_4) as in hydronephrosis and pyelonephritis or due to impaired excretion of ammonia in cases of renal failure.

Respiratory acidosis.

It is basically an increased carbon dioxide tension in the arterial blood. It may originate from :

— Airway obstruction : hypercarbia

— Insensitive or depressed respiratory centre : underventilation.

— Paresis of respiratory muscles.

— Carbon dioxide poisoning.

Respiratory acidosis is associated with increased arterial carbon dioxide tension and bicarbonate content. Metabolic acidosis is associated with reduced arterial carbon dioxide tension and bicarbonate content.

The clinical picture of acidosis is essentially the picture of the underlying cause. Patients with respiratory acidosis complain of respiratory discomfort while patients with metabolic acidosis do not complain of dyspnoea although Kussmaul breathing is present. Acidotic patients may show irregular jactitations and mental confusion followed by coma.

Treatment of acidosis includes :

— An attack to the cause :

In metabolic acidosis due to diabetic ketosis, give special diet and insulin. In respiratory acidosis, deal with the cause as respiratory obstruction.

— An attack to the acidosis :

Sodium bicarbonate or sodium lactate is given. Sixth molar sodium lactate is generally used. Molar sodium lactate is given when there is associated hyponatraemia. Sodium bicarbonate is used when there is a doubt about lactic acidosis. When it is essential to avoid sodium or potassium administration, anion exchange resins can be used orally.

AIKALOSIS

Basically, alkalosis is a condition of hydrion deficit. It may be metabolic or respiratory in origin.

Metabolic alkalosis is a condition of base excess or of a deficit of acids other than carbonic acid. In metabolic alkalosis, the renal response is a massive excretion of bicarbonate while the respiratory response is underventilation. In respiratory alkalosis, the expected picture is of hyperventilation or hypocapnea.

Metabolic alkalosis occurs during vomiting or gastric suction. Sodium or ammonium chloride are used for treatment. If vomiting or gastric suction are prolonged as in pyloric stenosis, there is depletion of sodium, potassium and chloride ions. Correction of potassium deficit may be important before treatment of the alkalosis itself. The actual treatment in such a situation is to remove or adjust the predisposing cause and to correct the fluid imbalance.

In hypokalaemic alkalosis, there is low concentration of potassium, raised bicarbonate in serum and a raised hydrogen ion concentration. Generally, the condition is a manifestation of potassium depletion. Not all cases of hypokalaemia manifest themselves by alkalosis. In primary alkalosis, there is potassium depletion. Patients receiving diuretics have low potassium levels and these diuretics are alkalinising by themselves. Potassium loss is associated with alkalosis, in the same way as sodium loss is associated with acidosis.

Regulation of acid-base balance :

Inspite of there being a constant stream of acidic and basic radicles set free into the blood stream, the hydrogen ion concentration (PH) of the blood remains remarkably constant ($\pm$ 0.1). This constancy is maintained through the contribution of the buffer systems, the lungs and the kidney.

Buffer systems :

A buffer is a solution which resists change of pH, when either acid or alkali is added to the blood. A buffer is usually a mixture of a weak acid and one of its salts. «weak» implies a slight degree of dissociation (ionisation) and «strong» means almost complete ionisation.

Buffering systems of the blood may be through the plasma or the red blood cells.

Exemples of plasma buffers :

$\frac{H_2\ CO_3}{B\ HCO_3}$; $\frac{B\ H_2PO_4}{B_2H\ PO_4}$ and $\frac{H\ Pr.}{B\ Pr}$, where, B, stands for base and Pr. stands for protein.

Exemples of red cell buffers :

$\frac{H.\ H\ BO2}{B.\ H\ BO2}$ and $\frac{H.\ HB}{B.\ HB}$, where B, stands for potassium as a base, and HB, stands for reduced haemoglobin.

In each system :

$pH = PK + Log \frac{Salt}{acid}$, where pK stands for the dissociation

constant of the particular acid and equals 6.1 while $\log \frac{NaHCO_3}{H_2CO_3} =$ 1.3. So, pH depends on the ratio of salt to acid.

Exemple : fixed acids as lactic, phosphoric and sulphuric acids are well ionized and react with bicarbonate of the plasma, thus :

HA (fixed strong acid) + B HCO3 $\rightarrow$ H2 CO3 + BA, where H2 Co3 is a weak acid. So, a strong acid which would reduce the pH + much, is converted to a less completely ionised or weak acid which affects the pH less and is unstable. The ratio $\frac{B\ H\ CO3}{H2\ CO3}$ would tend to be altered by this reaction and the pH affected, but, H2 CO3 being unstable easily gives CO2 which is eliminated by the lungs. There is also a reaction between carbonic acid and the amino group of haemoglobin molecule and so carbon dioxide combines and forms carbamino-haemoglobin.

The role of the lungs :

Excess carbon dioxide, from plasma bicarbonate and from metabolism is eliminated by pulmonary ventilation. The respiratory centre and the peripheral chemo-receptors are sensitive to carbon dioxide. In acidosis, carbonic acid is thus reduced and the ratio $\frac{Na\ HCO_3}{H_2\ CO_3}$ is kept normal. In alkalosis, pulmonary ventilation is decreased.

Role of the kidneys :

The kidneys share in regulation of acid — base equilibrium through :

— Excreting acid or base and maintaining the $\frac{BH_2PO_4}{H_2HPO_4}$ and $\frac{H_2CO_3}{BHCO_3}$ of the blood.

— Producing ammonia NH_3 (from enzymatic hydrolysis of glutamine) which combines with excess hydrogen ions to form NH_4, which is excreted in urine with available anions as chlorides and sulphate. So, excess hydrogen is excreted and in compensation sodium and other cations are re-absorbed. After an excessive loss of hydrogen ions, on the other hand, as occurs in severe vomiting, there is a corresponding decrease in ammonia production by the kidneys.

LABORATORY INVESTIGATIONS

Plasma sodium concentration ranges normally between 137 and 148 mEq/L. It is an index of osmolarity of E.C.F.

A low sodium concentration may indicate :

— Primary sodium deficit.

— Primary potassium deficit.

— Primary water excess

— A combination of these

So, a low sodium concentration is only a confirmatory evidence for a clinical diagnosis.

A high sodium concentration means :

— Primary aldosteronism, or

— Hypertonic saline solution administration.

Plasma potassium concentration ranges normally between 3.9 and 5.0 mEq/L.

Potassium concentration in the normal range does not exclude potassium depletion.

Levels below 3.5 m.Eq/L mean potassium depletion. Raised potassium concentration in a non-haemolysed sample denotes an urgent need for treatment to prevent cardio-toxicity.

Plasma chloride concentration ranges normally between 100 and 105 mEq/L. A low chloride concentration in relation to sodium concentration may denote metabolic alkalosis or respiratory acidosis. A high chloride concentration in relation to sodium concentration, may denote metabolic acidosis or respiratory alkalosis.

Plasma bicarbonate ranges normally between 22 and 20 mEq/L. It is low in metabolic acidosis and respiratory alkalosis and high in metabolic alkalosis and respiratory acidosis.

Regarding the hydrion, the normal pH of the blood ranges between 7.44 and 7.36. It is low in acidaemia with or without acidosis and high in alkalaemia with or without alkalosis. In respiratory alkalosis or acidosis, the changes in pH occur rapidly.

The clinical picture of the patient added to the estimation of bicarbonate and arterial carbon dioxide tension can differentiate between the respiratory and the metabolic components of a particular acid-base imbalance.

The Astrup interpolation technique for the investigation of the acid-base status of the blood

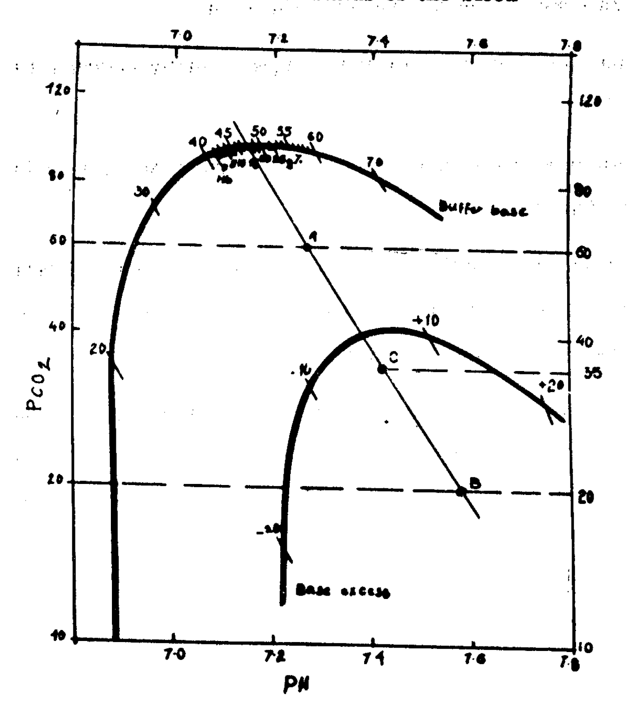


figure (1)

Principle of Astrup interpolation technique plotted on siggaard Andersen nomogram

The drawn blood has three unknowns, pH, $PaCO_2$ and HCO_3 which are determined by the Astrup-interpolation technique.

Blood sample is divided into three parts.

— Part I is used to measure the blood pH. The $PaCO_2$ and HCO_3 remain unknown.

— Part II is equilibrated with known low PCO_2 e.g. 28 mmHg, and the resulting pH is measured.

— Part III is equilibrated with known high PCO_2 e.g. 56 mmHg, and the resulting pH is measured.

The points described for part II and III are marked on a Sigaard-Anderson nomogram. This is a plot of pH (a log function of hydrogen ion concentration) on the abcissa against log PCO_2 on the ordinate. The conversion to log PCO_2 is achieved automatically because PCO_2 is expressed on a semi-log scale.

When the two points are joined by a straight line, the buffer line, it describes all the possible combinations of pH and PCO_2 that can exist in that particular blood.

The pH from part I is now referred to this line and the original PCO_2 is read off.

The remaining unknown is the bicarbonate.

Across the middle of the nomogram at $PCO_2 = 40$ mmHg, a horizontal line is marked in mEq/L for HCO_3. This line provides a computation of the Hendersen-Hasselbach equation in the form of :

$$pH = PK + Log \frac{BHCO_3}{H_2CO_3} \quad \text{or}$$

$$HCO_3 = \text{antilog} (PH - PK + \text{Log } PCO_2 + \log 0.03)$$

When a line from the point which describes part I is drawn at 45° so that it intersects this bicarbonate line, the intercept gives the answer to the computation and the «actual bicarbonate» is read off.

The point where the buffer line intersects the bicarbonate line is the «Standard bicarbonate», i.e. where $PCO_2 = 40$ mmHg which is the definition of standard bicarbonate.

Where the buffer line intersects a lower «base excess» curve, the base excess of the sample can be read off. This enables the investigator to calculate the amount of bicarbonate necessary to remedy the metabolic component of the blood.

Where the buffer line intersects a higher «buffer base» curve, is the value of the total bases of the blood including haemoglobin which can also be determined from the nomogram.

Dose of bicarbonate in mEq. = base deficit × 0.3 × patient weight in kilograms.

THE IMMUNE RESPONSE

The defensive mechanisms of the human body include non specific suppressive mechanisms and specific immune responses. The ciliary action of the tracheobronchial tree and the lytic action of fatty acids in the sweat and sebaceous secretions are exemples of the non-specific suppressive mechanisms.

The immune response is a complex series of immunolagical reactions which identify immunogens (foreign agents) present in the organism and try to make them less harmful or harmless.

Lymphocytes are the main cells responsible for the immune response but other cells also contribute.

The concept of two-component immune system implicates two different lymphocyte populations ; the T- and B- lymphocytes. T-lymphocytes are thymus derived and are responsible for cell-mediated immunity. B-lymphocytes are thymus-independent and and are responsible for humoral immunity.

The cell-mediated immune response is responsible for delayed hypersensitivity reaction, graft versus host reaction, the rejection of tissue transplants and tumour cells and the prevention of certain infections. T-lymphocytes are the responding cells and they identify the antigens. They are activated and transformed into lymphoblasts. They can synthesise and release several biologically active, soluble, non-antigen-specific factors (lymphokines) which are responsible for different functions as chemotaxis. Activated T cells may also produce the macrophage arming factor (antigen receptor of the T-cells or part of it), which attaches to the surface of macrophages and stimulates them to specifically kill target cells that have the same surface antigen structure as the stimulating cells. The activated cells are further transformed, possibly in cooperation with macrophages, into memory cells and killer cells, which are able to destroy the cell carrying the antigen.

In the humoral immune response, B-lymphocytes are activated and transformed into antibody — producing plasma cells. Antibodies circulate with gamma-globulins as immunoglobulins including 5 types. The presence of T-lymphocytes is necessary for optimal antibody synthesis. They act as helper or suppressor cells when macrophages also participate. Thus macrophages treat and concentrate the antigen and deliver it to the B-lymphocytes.

Macrophages are the third type of cells involved in the function and control of immune reactivity.

Other factors are :

— The nature of the antigen.

— The vehicle in which the antigen is introduced

— The root and amount of antigen.

— The presence in the host of antigens or antibodies homologous or cross-reactive with that antigen.

Thus the expression of immune response in a host to a given antigen reflects an interplay of cellular and humoral components. The complement system is formed of plasma enzymes which also mediate the antigen antibody reaction.

How do lymphocytes identify the antigens ? This is achieved by receptors on the surface of lymphocytes. T-lymphocytes receptors have not yet been identified, while B-cell receptors have been identified as immunoglobulin molecules.

The receptors and their capacity to synthesize specific antibodies are genetically controlled.

The antigen must have a sufficiently high molecular weight in order to be identified. A small molecule may be attached to a larger protein antigen acting as a carrier, therefore creating an immune response which is specific to the small molecule antigen. In that situation, identification apparently requires cooperation between T and B-lymphocytes. B-lymphocytes identify the small molecule antigen while T-lymphocytes identify the carrier protein.

The first exposure of the antigen to the organism creates a reaction termed the primary immune response. The memory cells developed from both T and B-lymphocytes, which have retained the information concerning the antigen, respond rapidly upon encountering the same antigen again when a secondary immune response develops. This response shows a reaction which is more intense than the primary reaction and proliferation of genetically — programmed cell clones take place.

The immune response is an event in which the participating cells cooperate directly or through soluble products. Factors which stimulate or inhibit this process at some stage, may improve or impair the ability of the body to react against infection, tumour cells or foreign tissues.

The clonal selection theory of antibody production dictates that the genetic information governing the making of each antibody is stored within the genome of each cell. The appropriate cell must recognize an antigen to which it is previously committed before it begins to synthesize the corresponding antibody. After recognizing an antigen, the cell proliferates and gives rise to a

clone of cells, all committed to the same antigen. Of these, some proceed to differentiate into plasma cells producing the same antibody. Other cells remain as antigen-sensitive precursor cells, now present in lager numbers, ready to respond again to the antigen and thus forming the basis for the secondary response or immunological memory.

The strongest prediction of the theory is that each antigen-sensitive cell is pre-committed to respond to a single antigen specificity despite the fact that the cell genome holds information for a wide range of responses. So, the antigen recognition takes place on the surface of the cell that will give rise to the specific antibody producing cells for that antigen. So, the antigen-sensitive lymphocytes (B-cells) display on its surface a «sample» of the antibody to which it is committed and this serves as a receptor to detect the presence of the appropriate antigen in the environment.

The immune response requires the intimate interaction of three or more cell types : B-cells, macrophages and T-cells. Macrophages localize and concentrate antigen and perform limited degradation, making it possible to present the antigen in a useful form to lymphocytes. The existence of lymphocyte factors that influence macrophage behaviour, i.e. migration inhibition factor, is well known.

The subunit structure of immunoglobulins consists of 4 polypeptide chains which are : 2 identical light chains and 2 identical heavy chains Each chain has 2 binding sites : one for binding

the antigen and the other for binding the complement Immunoglobulins are classified into 5 major classes. Distinction is based on physico-chemical and antigenic differences of heavy chains. These classes are Ig A, Ig D, Ig E, Ig G and Ig M. They are further subdivided into subclasses. In normal human serum, Ig G accounts for 85% of the total. Ig G is the only member which crosses the placental barrier and gives neonatal immunity. Ig A provides protection in exocrine secretions. Ig E provides reaginic activity. Ig M fixes complement more efficiently than others Ig D biological function is not yet determined.

Assessment of immunological responses, in vitro, is achieved through different ways. Antibodies may be assayed by binding with radioactive antigen or visualised microscopically after labelling with fluorescent compounds. Tests of cell-mediated immune function are the lymphocyte transformation test and the leucocyte migration inhibition test. Both depend on the in vitro recognition of antigen by sensitized lymphocytes.

It is stressed that the specific immune responses and non-specific resistance mechanisms are intimately inter-related. For exemple, the attachement of specific antibody to bacterial wall promotes phagocytosis by macrophages. Then, «Processing» of antigen by macrophages and subsequent presentation to lymphocytes, stimulates more active specific B and T cells involvement. Antigen-antibody complexes, by activating the complement system, promote phagocytosis. Lysis of bacteria and neutralization of toxins and the phagocytosis of virus particles may be encouraged by their initial nentralization by serum antibody.

ANAESTHESIA AND THE IMMUNE RESPONSE

The effect of anaesthesia on the immune response can be discused under four titles :

1. Leucocytic and lymphocytic counts.
2. Lymphocyte transformation.
3. Effect on antibodies.
4. Clinical implications.

In most of the situations the effect of operative surgery overshadows the effect of anaesthesia. Studies of the effects of anaesthetics in vitro are purely descriptive to anaesthesia, but it is not yet proved that invitro studies are mirror image of those occuring in vivo. Laboratory studies of experimental animals helped in pointing out anaesthetic effects.

1. LEUCOCYTIC AND LYMPHOCYTIC COUNTS

Since the start of the present century, ether anaesthesia was shown to increase neutrophilic leucocytes and lymphocytes in peripheral blood of both man and laboratory animals. Halothane anaesthesia of long duration causes leucopenia characterized by neutrophilia and lymphopenia in the rat. Nitrous oxide anaesthesia induces leucopenia in the rat also. Neutropenia and leucopenia were reported in animals after barbiturate anaesthesia.

Results of anaesthesia and surgery in man are conflicting. But on the whole they agree with the effects of anaesthesia in the

laboratory animal. While general anaesthesia was followed by leucocytosis, regional anaesthesia brought about no leucocytosis.

Some authors demonstrated a decrease of both T- and B-lymphocytes in peripheral blood during and immediately after surgery. Others reported selective decrease of T-lymphocytes and a relative increase of B-lymphocytes.

2. LYMPHOCYTE TRANSFORMATION

Halothane, in concentrations used for clinical anaesthesia, was found to inhibit the DNA synthesis of human lymphocytes, but the time of action required for such an invitro effect was several days. On the other hand, nitrous oxide, thiopentone and ketamine, in clinical concentrations, did not affect lymphocyte transformation in vitro. Halothane was found to suppress the function of T-lymphocytes in the chicken.

In clinical practice some authors reported that barbiturate anaesthesia (thiopentone), combined general anaesthesia (thiopentone, nitrous oxide, pethidine) and combined halothane anaesthesia (thiopentone, nitrous oxide, halothane) did not affect lymphocyte transformation. Other investigators reported various degrees of depression of lymphocyte transformation response, correlated with the degree of the surgical trauma but not with the duration of the operation or the anaesthetic agents employed. Blood loss has been found to correlate with the depression of the lymphocyte transformation.

In addition, the effect of anaesthesia and surgery on immunity was modified by the effects of hormonal changes caused by anaesthetic drugs, immunological results of the stress response to exposure to anaesthesia and surgery, the use of antibiotics, steroids and the coincidental viral infections.

3 — EFFECT ON ANTIBODIES

Gammaglobulin concentration has been found to decline transiently in man during the first few post operative days. Serum Ig G, in adults, was found to decline during the first post operative week, in correlation with the degree of trauma. The literature is very poor as to the effect of anaesthesia on antibody production in man.

4. CLINICAL IMPLICATIONS

Anaesthetic drugs are known to suppress non-specific immune mechanisms of the body. For exemple, inhibition of the ciliary action in the tracheobronchial tree by halothane, nitrous-oxide or pancuronium allows the accumulation of bronchial secretions. Again they were shown by some authors to depress the specific immune response in vitro, in the experimental animal and sometimes in man. Different mechanisms of immunosuppression were suggested including decreased motility and phagocytic power of macrophages, inhibition of lymphocyte stimulation and transformation and inhibition of cell division by inhibiting DNA synthesis.

The inhibition of the immune response due to anaesthesia or anaesthesia and operative surgery may increase the incidence of infection of surgical patients. It is also possible that the prolonged exposure of theatre personnel to sub-anaesthetic doses of anaesthetic vapours and gases makes them more susceptible to infection. In a recent study a higher incidence of malignancy was reported in anaesthetists than pediatricians, the difference being statistically significant. This may be due to depression of their cellular immunity. In addition, cellular immunity has been blamed responsible for the rejection of organ transplants.

Lastly, it would be better to clarify some confusing terminology. True «allergy» or «hypersensitivity» to a drug implies the production of specific antibodies and/or sensitized lymphocytes which can be demonstrated. If immunological responses cannot be demonstrated, «intolerance» or «idiosyncrasy» are the descriptive terms. Some intravenous anaesthetics cause histamine relase, and this is not a true «allergic» reaction as it is a pharmacological rather than an immunological response. But owing to the similarity of such a reaction with antibody-mediated anaphylaxis, it can be described as an «anaphylactoid» reaction.

THE SYSTEM INTERNATIONAL (S.I.)
OF MEASUREMENT

The following informations on S.I. units and factors for conversion between S.I. and older conversional units is provided for the convenience of the anaesthetist. This system allows unified interchange of knowledge between all scientific centres in the world. The anaesthetist will need to consult this system every now and then.

Basic SI Units

Physical quantity	Name	Symbol
Length	Metre	m
Mass	Kilogram	kg
Time	Second*	s
Electric current	Ampere	A
Thermodynamic temperature	Kelvin	K
Luminous intensity	Candela	cd
Amount of substance	Mole	mol

* Minute (min), hour (h) and day (d) will remain in use although they are not official SI units.

Prefixes for Si Units

Factor	Name	Symbol	Factor	Name	Symbol
10^{18}	Exa	E	10^{-18}	Atto-	a
10^{15}	Peta	P	10^{-15}	Femto	f
10^{12}	Tera-	T	10^{-12}	Pico-	p
10^{9}	Giga-	G	10^{-9}	Nano-	n
10^{6}	Mega-	M	10^{-6}	Micro-	u
10^{3}	Kilo-	k	10^{-3}	Milli-	m
10^{2}	Hecto-	h	10^{-2}	Centi-	c
10^{1}	Deca-	da	10^{-1}	Deci-	d

DERIVED SI UNITS

Quantity	SI unit	Symbol	Expression in terms of SI base units or derived units
Frequency	Hertz	Hz	1 Hz = 1 cycle/s (1 s^{-1})
Force	Newton	N	1 N = 1 kg. m/s^2 (1 kg. mps^{-2})
Work, energy, quantity of heat	Jolue	J	1 J = 1 N. m
Power	Watt	W	1 W = 1 J/s (1 $J.s^{-1}$)
Quantity of electricity	Coulomb	C	1 C = 1 A. s
Electric potential, potential difference, tension, electromotive force	Volt	V	1 V = 1 W/A (1 W. A^{-1})
Electric capacitance	Farad	F	1 F = 1 A. s/V (1 A. s. V^{-1})
Electric resistance	Ohm	Ω	1 Ω = 1 V/A (1 V. A^{-1})

(Suit)

Quantity	SI unit	Symbol	Expression in terms of SI base units or derived units
Flux of magnetic induction, magnetic flux	Weber	Wb	$1\ Wb = 1\ V.\ s$
Magnetic flux density, magnetic induction	Tesla	T	$1\ T = 1\ Wb/m^2\ (1\ Wb.\ m^{-2})$
Inductance	Henry	H	$1\ H = 1\ V.\ s/A\ (1\ V.\ s.\ A^{-1})$
Pressure	Pascal	Pa	$1\ Pa = 1\ N/m^2\ (1\ N.\ m^{-2})$
			$= 1\ kg/m.\ s^2\ (1\ kg.\ m^{-1}.\ s^{-2})$

The litre ($10^{-3}\ m^3 = dm^3$), though not official, will remain in use as a unit of volume as also will the dyne (dyn) as a unit of force ($1\ dyn = 10^{-5}\ N$).

SI unit	Old Unit	Conversion factors	
		Old to SI (exact)	SI to old (approx.)
kPa	mm Hg*	0.133	7.5
kPa	1 standard atmosphere** (approx : 1 Bar)	101.3	0.01
kPa	cmH_2O	0.0981	10
kPa	Ibs/sq in	0.145	7

* e.g. systolic BP of 120 mmHg = 16 kPa and diastolic BP of 80 mmHg = 11 kPa.

** = 760 mmHg.

Measurement	SI unit	Old unit	Conversion factors: Old to SI (exact)	Conversion factors: SI to old (approx.)
● **Blood**				
Acid-Base				
PCO_2	kPa	mmHg	0.133	7.5
PO_2	kPa	mmHg	0.133	7.5
Standard bicarbonate	mmol/litre	mEq/litre	Numerically equivalent	
Base excess	mmol/litre	mEq/litre	Numerically equivalent	
Glucose	mmol/litre	mg/100 ml	0.0555	18
● Plasma				
Sodium	mmol/litre	mEq/litre	Numerically equivalent	
Potassium	mmol/litre	mEq/litre	Numerically equivalent	
Magnesium	mmol/litre	mEq/litre	0.5	2
Chloride	mmol/litre	mEq/litre	Numerically equivalent	
Phosphate (inorganic)	mmol/litre	mEq/litre	0.323	3.0
Creatinine	umol/litre	mg/100 ml	88.4	0.01
Urea	mmol/litre	mg/100 ml	0.166	6.0
● Serum				
Calcium	mmol/litre	mg/100 ml	0.25	4.0
Iron	umol/litre	ug/100 mol	0.179	5.6
Bilirubin	umol/litre	mg/100 ml	17.1	0.06
Cholesterol	mmol/litre	mg/100 ml	0.0259	39
Total proteins	g/litre	g/100 ml	10.0	0.1
Albumin	g/litre	g/100 ml	10.0	0.1
Globulin	g/litre	g/100 ml	10.0	0.1

Biochemical-Content of Other Body Fluids

Measurement	SI unit	Old unit	Conversion factors: Old to SI (exact)	Conversion factors: SI to old (approx.)
● Urine				
Calcium	mmol/24 h	mg/24 h	0.025	40
Creatinine	mmol/24 h	mg/24 h	0.00884	113
Potassium	mmol/litre	mEq/litre	Numerically equivalent	
Sodium	mmol/litre	mEq/litre	Numerically equivalent	
● Cerebro-spinal fluid				
Protein	g/litre	mg/100 ml	0.01	100
Glucose	mmol/litre	mg/100 ml	0.0555	18

Haematology

Measurement	SI units	Old unit	Conversion factors: Old to SI	Conversion factors: SI to old
Haemoglobin (Hb)	g/dl	g/100 ml	Numerically equivalent	
Packed cell volume	No unit*	Per cent	0.01	100
Mean cell Hb conc.	g/dl	Per cent	Numerically equivalent	
Mean cell Hb	pg	uug	Numerically equivalent	
Red cell count	Cells/litre	Cells/mm^3	10^6	10^{-6}
White cell count	Cells litre	Cells/mm^3	10^6	10^{-6}
Reticulocytes	Per cent	Per cent	Numerically equivalent	
Platelets	Cells/litre	Cells/mm^3	10^6	10^{-6}

* Expressed as decimal fraction, e.g. normal adult male value 0.40 to 0.54.

pH and Nmol/Litre of

H+ Activity

pH	mmol/litre
6.80	158
6.90	126
7.00	100
7.10	79
7.20	63
7.25	56
7.30	50
7.30	45
7.40	40
7.45	36
7.50	32
7.55	28
7.60	25
7.70	20

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